Peter von Theobald
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Editors

New Techniques in Genital Prolapse Surgery

John Lumley
Nadey Hakim *Series Editors*

Springer

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Preface

This new book aims to interest gynecologists, urogynecologists, urologists, colorectal surgeons, and all other pelvic surgeons concerned with surgical treatment of prolapse. Approximately 11% of women have undergone surgery for a genital prolapse by the age of 80. Genital prolapse operations are among the most common and frequent operations in women after hysterectomy and c-section. As life expectancy increases and as patients demand a higher quality of life, the number of patients (and surgeons) concerned with this issue is growing.

Many of the common techniques for prolapse repair are rather unchanged since the end of the nineteenth century when most of the techniques were established. Colpectomy, colporrhaphy, perineorrhaphy, hysterectomy, fascial repair, and myorrhaphy are still the most frequent techniques used in routine surgery. These procedures depend heavily on anatomically distorting plication for bulge reduction.

New techniques appeared in the 1950s and 1960s. They include abdominal sacral colpopexy using mesh by Scali, the sacrospinous ligament fixation by Richter, and the McCall culdoplasty procedures. These "new" techniques were aiming to restore apical support that was not possible with any of the "plication" techniques. Even today, only trained surgeons are able to perform them routinely because of poor reproducibility and perceived complexity.

A revolution occurred with stress urinary incontinence (SUI) surgery in the late 1990s when Ulmsten and Petros started using synthetic meshes as suburethral slings by the vaginal approach with very low complication rates. Compared to the traditional procedures, this new technique was much easier to perform, less invasive, less morbid, standardized, and more efficient. The initial surge of the TVT procedure was in Europe between 1998 and 2000. In France, Italy, and Belgium, 100% of SUI operations were performed with this technique from 2001 until the technique was changed to the transobturator sling between 2002 and 2004. The changes were slower to come in the UK and the USA, but now these mesh techniques have largely replaced older forms of urethropexy.

Application of mesh to surgery for prolapse repair was a logical consequence of the success of TVT SUI surgery. Mesh procedures started becoming popular with the new millennium and aimed to be less invasive and more efficient than the traditional techniques. After ten years of evolution, standardized techniques have emerged for cystocele repair, vault prolapse suspension, and enterocele and rectocele repair. A high degree of interest for these new techniques is shown by all pelvic floor surgeons, whether they are innovators, already using these techniques, or more conservative and showing allegiance to the traditional placation techniques. In any gynecologic or urologic surgery congress including a session about mesh repair, the room is full. Surgeons want information about mechanically superior, anatomically restorative pelvic organ prolapse procedures. Many of the papers published in the concerned journals are on this subject, but, to date, no book has been published specifically addressing this topic. We believe that it is timely now for a well-documented book containing simple, practical, and useful information written by international experts in this field. This book could become the "bible" of the new genital prolapse surgery using mesh.

To label a book *New Techniques in Genital Prolapse Surgery* may seem ambitious; however, the goal of the editors and authors is to prove that the title is indeed accurate. Definitely, many concepts and techniques are new surgery replacing old and biomechanically inferior procedures.

First, anatomy is seen differently. Fixed anatomy, as depicted in drawings or dissections, is now viewed as mobile, dynamic, and functional. The mobility of the organs and the modifications of the axes during rest or straining are considered key points to understanding the pathogenesis of pelvic floor defects and their repair. Balanced forces acting in opposite directions at different levels with low or no tension provide a physiologic support mechanism and hammock that suspends the pelvic floor without rigidity.

Second, the philosophy of repair is new. The new surgery aims to create new connective tissue to replace broken ligaments and septa instead of trying to tighten or to suture an altered suspensory apparatus. Synthetic meshes or biologic grafts are used for this purpose. Thus, vaginal or vulvar narrowing techniques, extensive ligamentoplasty, or deep myorrhaphies are now becoming of historical interest only. Anatomy is restored rather than distorted, and postoperative pain is tremendously reduced.

Third, new pathways for fixations are used, like the infracoccygeal translevatoric approach for vault prolapse or the double transobturator approach for cystocele. Surgical technique is simplified and becomes more reproducible and quicker.

Fourth, a new field of research has been opened concerning prostheses and biomaterials. Early in the development of this surgery, hernia meshes were used. Now specifically designed meshes for prolapse surgery are produced by engineers to meet the specific needs of surgeons. Our knowledge about vaginal foreign body reaction has dramatically changed, and we are only at the start of this new era!

Most of the authors of this book started applying this new surgery in 2000 or 2001 and tried from the start to standardize the different procedures and to evaluate and improve the different grafts. This book is aiming to describe the state of art in terms of knowledge, techniques, results, and complications management.

If the 1980s and 1990s were the years of the laparoscopic surgery revolution, the new millennium has started with the urogynecologic surgery revolution, but there is a difference. If laparoscopic surgery was trying to mimic the same techniques as traditional surgery through a new approach, the new prolapse surgery is trying to modify dramatically the traditional techniques through the same incision!

We believe "New Techniques in Genital Prolapse Surgery" will be the first reference book for the new generation of pelvic reconstructive surgeons that will use mesh as a routine procedure via safe and effective means.

Peter von Theobald
Carl W. Zimmerman
G. Willy Davila

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Part I

Anatomy and Function

New Considerations About Pelvic Floor Anatomy

1

Carl W. Zimmerman

Introduction

Historically, the science of anatomy has been descriptive in nature. Gross and microscopic observations have been progressively taken to an increasingly reductionist level in the modern era. Since the advent of surgical procedures, anatomy of the living[1] has emerged as a distinct set of observations that are very different from those seen in the fresh or fixed cadaver. More recently, imaging with the use of radiation, magnetic, and ultrasound energy has progressed to the point that some aspects of anatomy can now be observed in normal living individuals without surgical entry into the body or tissue plane dissection.[2-6] As a result of improved imaging techniques, dynamic changes in anatomical relationships can now be studied under normal and various anatomically altered circumstances. No part of the human body is more amenable to this type of dynamic analysis than the female pelvis. An understanding of normal female pelvic anatomy requires a biomechanical analysis of the forces that constantly act upon the pelvic floor and the structures that resist those forces.

The normal functional actions of the central pelvic structures are micturition, defecation, coitus, and parturition. All of these functions involve changes in anatomy from the normal resting state. Some of these changes are more subtle and some are more dramatic. In the particular example of childbirth, significant stress is exerted on the connective tissue elements of the endopelvic fasciae creating unavoidable damage that can potentially impact pelvic organ support, suspension, and function for the remainder of a women's life. No longer are static descriptions of the interrelationships of structures sufficient to understand the pathophysiology and treatment of altered pelvic anatomy. In this chapter, emphasis will be placed on creating a synthesis between traditional descriptive anatomy and emerging biomechanical concepts to create a more complete understanding of the alterations that occur during and after the development of pelvic organ prolapse.

Support of the Central Pelvic Organs

Support of the uterus, vagina, bladder, and rectum is furnished by the bony pelvic girdle and the pelvic diaphragm composed of the levator ani muscles.[7] The large central defect in the bony pelvis is partially occluded by these highly adapted muscles of the pelvic floor. This occlusion of the pelvic outlet is not complete. The urogenital hiatus is a central defect in the muscular floor of the pelvis that allows for coitus, childbirth, and the elimination of bowel and urinary waste. Several evolutionary adaptations have developed in the female human pelvis that reduces the impact of gravitational and physiological forces on the pelvic floor (Table 1.1). Each of these adaptations will be discussed in turn.[8]

Lumbosacral lordosis is a result of a gentle ventral facing curve in the lumbar and sacral spine that is especially pronounced in reproductive aged females. This curvature is completed by the posterior tilt of the sacrum with the central portion of the arc located at L5-S1. The effect of this curvature is that the pelvic inlet is in a nearly vertical position similar to that of a quadruped. The anterior deviation of the sacral promontory essentially places it in a vertical plane over the pubic symphysis. In the standing position, gravitational forces are deflected onto the anterior portion of the pelvic girdle rather than directly on the pelvic outlet. With increasing age, lordosis is gradually replaced by kyphosis. As a result, gravitational forces are directly absorbed by the pelvic floor making the development of prolapse more likely.[9]

Humans are the only species with a substantial degree of anterior concavity of the sacrum.[10] This arc continues into the rudimentary coccygeal portion of the spinal column. This

Table 1.1 Evolutionary adaptations affecting the structure and function of the female human pelvis

Lumbosacral lordosis
Internally concave sacrum
Ischial spines
Coccygeal regression with ventral deviation
Levator ani modification

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adaptation reduces the size of the pelvic outlet and allows adapted muscular and connective tissue elements to form the uniquely hominid placement of the urogenital hiatus in a dependent position.[11] Attached to the anterior pointing tip of the coccyx is a dense aponeurosis of connective tissue named the sacrococcygeal raphe. This structure is sometimes referred to as the levator plate and is an integral part of the adaptations of the levator ani muscles. This firm connective tissue aponeurosis bears much of the downward force exerted against the dependant pelvic floor.[12]

Ischial spines are also unique human structures.[13,14] These bilateral protuberances of bone arise from the ischium between the greater and lesser sciatic notches and create a posterior mid-pelvic plane of narrow dimension named the interspinous diameter. This restriction of the bony mid-pelvis and the internally concave sacral curve causes the term infant to undergo the cardinal movements of labor known as flexion, descent, internal rotation, and extension. These maneuvers place specific and predictable force vectors on the deep endopelvic connective tissue (endopelvic fasciae) and the musculature of the pelvic floor. In fact, all the named components of the deep endopelvic connective tissue intersect within the interspinous diameter converging to form the pericervical ring. Because this plane is the narrowest diameter of the pelvis, the highest pressures generated within the pelvis during labor are located in this area. The result of the process is avulsion and displacement of the endopelvic fasciae away from the interspinous diameter. This damage to the continuity of the named elements of the endopelvic fasciae compromises suspension of the central pelvic organs above urogenital hiatus of the pelvic diaphragm.[15]

Coccygeal regression is coupled with anterior displacement of this rudimentary structure to help occlude the bony pelvic outlet. The sacrum, coccyx, and sacrococcygeal raphe or aponeurosis serves as attachment sites for the muscles of the pelvic diaphragm. These paired muscles serve the function of the pelvic support.

In primates other than humans, the levator ani muscles are primarily responsible for movement of the elongated tail. These same muscles in the human coupled with sacrococcygeal regression and central insertion of the levator ani muscles help retain abdominal and pelvic contents within the abdomen.

The muscles of the pelvic diaphragm along with the bony pelvic girdle help to furnish support to the structures of the female pelvis. These muscles are also known as the levator ani. These skeletal muscles have a parietal fascia superiorly (superior fascia of the pelvic diaphragm) and inferiorly (inferior fascia of the pelvic diaphragm.) These paired muscles arise from the bones of the pelvic girdle and medial fascia of the obturator internus muscle. They insert medially onto the lateral sacrum, coccyx, and sacrococcygeal raphe. Central to the anterior and medial portion of the diaphragm is the urogenital hiatus that allows for the transit functions of the urinary, bowel, and reproductive tracts. Because this hiatus must be large enough to accommodate childbirth, it also permits development of various types of pelvic organ prolapse.[16]

Suspension of the Central Pelvic Organs

The function of suspension of the uterovaginal complex, bladder, and lower gastrointestinal tract is provided by the deep endopelvic connective tissue or endopelvic fascia. Histologically, this irregularly shaped structure is the fibroelastic connective tissue with varying degrees of smooth muscle and a significant elastic component.[17,18] Structurally, the endopelvic fascia is a continuation of the subperitoneal connective tissue that gradually becomes denser as one progresses from the respiratory diaphragm to the pelvic diaphragm. The endopelvic fascia is located within the space between the dependent portion of the pelvic peritoneum and the muscles of the pelvic diaphragm. At various locations this tissue condenses into paired ligament-like structures, septa that separate the vagina from the bladder and rectum, and a single pericervical ring located within the interspinous diameter (Table 1.2).

The uterosacral ligaments provide the primary apical suspensory function for the uterus, vagina, and their surrounding structures.[19] These ligaments are dense, highly collagenized, cable-like structures that arise from the presacral periosteum of S2–4 and parietal fascia of the piriformis muscle and insert onto the posterior and lateral cervix. In normal intact female pelvic anatomy, they hold the cervix posterior to the urogenital hiatus allowing it to rest on the support of the sacrococcygeal raphe. A significant autonomic nervous plexus is embedded within the uterosacral ligaments.

The cardinal ligaments have some functional significance in side-to-side stabilization and in suspension of the cervix. These structures are very similar to the mesenteries of the upper abdomen. They are a continuation of the hypogastric root.[20] They arise from a broad portion of the pelvic sidewall and insert on the lateral supravaginal cervix. The ureters and the uterine arteries and veins travel through these structures. The cardinal and uterosacral ligaments are continuous with one another; however, their functions are somewhat different.

Table 1.2 Named components of the endopelvic fascia

Uterosacral ligaments
Cardinal ligaments
Pubourethral ligaments
Pubocervical septum
Rectovaginal septum
Pericervical ring

The pubourethral or pubocervical ligaments arise from the pubic bone and insert onto the anterior cervix. They serve a minor role in stabilization of the cervix because they are not a part of the suspensory axes of the vagina. These structures are also known as the surgical bladder pillars and must be divided during hysterectomy. The paired uterosacral, cardinal, and pubourethral ligaments are roughly equilaterally placed around the cervix and along with the pericervical ring are collectively known as the paracolpium.

The trapezoidal pubocervical septum separates the dependent portion of the bladder from the epithelium of the vagina. It is part of the anterior arm of the suspensory axis of the vagina and supports the bladder. The pubocervical septum extends from its distal junction with the urogenital diaphragm to the anterior pericervical ring between the insertions of the pubourethral ligaments. Laterally, the pubocervical septum attaches to the medial fascia of the obturator internus muscle via the arcus tendineus fascia pelvis or white line.

The trapezoidal rectovaginal septum separates the anterior rectum from the posterior vaginal wall. It forms an integral part of the primary posterior suspensory axis of the uterovaginal complex. It extends from a distal junction with the perineal body to its apical termination at the pericervical ring between the uterosacral ligaments. It is a thicker and more substantial structure than the pubocervical septum because of its load bearing function. Its length is greater than that of the pubocervical septum by a distance equal to the diameter of the cervix. If the cervix is absent a connective tissue defect is created that cannot be surgically corrected in a completely anatomical way and a disruption between the anterior arm of the suspensory axis and the primary posterior arm occurs.

In intact female pelvic anatomy, all of the named components of the endopelvic fascia mentioned above converge within the interspinous diameter to form the pericervical ring. This structure encircles and stabilizes the supravaginal portion of the cervix. The net result is that the cervix is suspended in the posterior midpelvis by an integrated continuum of connective tissue condensations.[21] This area of stabilization is normally located posterior to the urogenital hiatus where the cervix rests on the dense medial convergence of the pelvic diaphragm, the sacrococcygeal raphe. All named components of the endopelvic fascia converge within the interspinous diameter to form the pericervical ring. The convergence of named structures within the interspinous diameter is the single most important integrative concept for the pelvic reconstructive surgeon. Restoration of structural connections within the interspinous diameter should be the primary goal of prolapse surgery.

A unique characteristic of the deep endopelvic connective tissue is the surgeon's ability to separate the named components from each other and from surrounding structures by way of avascular spaces.[22] If properly identified and dissected, these spaces allow the surgeon to gain access to the areas of critical importance to the reconstruction of the central pelvic soft tissues. A total of ten avascular spaces exist in the pelvis (see Table 1.3).

Table 1.3 Avascular spaces of the pelvis

Vesicovaginal
Vesicocervical
Vesicouterine
Prevesical
Paravesical[2]
Rectovaginal
Pararectal[2]
Retrorectal

Surgical Access to Pelvic Structures

Anterior pelvic reconstruction requires complete dissection of the vesicovaginal space. This space extends from the urogenital diaphragm to the interspinous diameter and laterally to the pelvic sidewall. Surgical development of this space allows access to the pubocervical septum.[23] Cystoceles are a result of an apical transverse separation of this structure from the pericervical ring. Paravaginal defects are a result of separation of the pubocervical septum away from the arcus tendineus fascia pelvis. Paravaginal defects create an abnormal connection between the vesicovaginal space and the fat filled ipsilateral paravesical space. No fat exists within the perivaginal structures. If the surgeon identifies fat during the dissection of an avascular space, a pelvic hernia has been identified. If the pubocervical septum is not disrupted on two contiguous sides, anterior vaginal prolapse cannot develop. Biomechanically, when a cystocele is present, at least one paravaginal defect is also present. Both of these contiguous defects must be repaired to reestablish the integrity of the anterior vaginal anatomy. The cardinal movements of labor above and within the interspinous diameter usually result in a unilateral right paravaginal defect and apical transverse defect creating both a central cystocele apical transverse defect and a unilateral (usually patient right) paravaginal defect. The mechanical failure of the anterior vaginal wall is then due to a flap-like defect in the pubocervical septum with displacement of the septum away from the right ischial spine. In a minority of cases, a unilateral left paravaginal defect and apical transverse defect are present. Bilateral paravaginal defects with an apical transverse defect may also be encountered, especially with complete degrees of prolapse.

The vesicocervical and vesicouterine spaces are important during hysterectomy and allow access to the anterior peritoneal fold during hysterectomy. The anterior portion of the pericervical ring separates the vesicocervical and vesicouterine spaces and is sometimes identified as the supravaginal septum.

The prevesical space is located anterior to the bladder. It is filled with fat and a plexus of veins. The lateral recesses of this space are called the paravesical spaces. These spaces are important during anterior urethropexy and abdominal paravaginal repair.

The rectovaginal space extends from the perineal body to the posterior pericervical ring. The lateral boundaries are the arcus tendineus fascia pelvis in the apical two thirds of the vagina and the arcus tendineus fascia rectovaginalis in the distal one third of the vagina. Surgical dissection of the rectovaginal space allows access to the rectovaginal septum. Childbirth related disruption of the rectovaginal fascia is much more constant and predictable than the variable presentations of anterior pubocervical fascia damage. During transit through the interspinous diameter, infants are normally oriented in the occipitoanterior position. With continued descent, the baby extends its head to follow the internally concave surface of the sacrum. The rectovaginal septum is placed under significant stress by the resulting force and is commonly separated from the posterior pericervical ring. This separation creates an apical transverse defect in the rectovaginal septum. The septum is displaced distally toward the perineum and allows rectocele and enterocele to develop through the same fascial defect.

Paired pararectal spaces are located apical to the ischial spines and lateral to the normal attachment site of the uterosacral ligaments to the pericervical ring. These fat-filled spaces allow surgical access to the mechanically intact retroperitoneal portion of the uterosacral ligaments. Surgical manipulation of these ligaments within the ventral portion of the pararectal space is useful for uterosacral colpopexy. Two to three centimeters of dense connective tissue separates this portion of the uterosacral ligament and the closest portion of the ureter. The sacrospinous ligament forms the inferior border of the pararectal space, and the coccygeus muscle form the posterior boundary.

Gynecological surgeons rarely use the retrorectal space. It is located posterior to the pelvic portion of the rectum and is sometimes accessed during operations for anal intussusceptions.

Biodynamics of the Functional Pelvis

The net effect of fascial and muscular pelvic support of the pelvic diaphragm and suspensory function of the endopelvic fascia is that the central pelvic organs are effectively suspended above the urogenital hiatus. In anatomically intact females, the resistance to descent is considerable as shown by Mengert in 1936.[19] At least two methods have been used to describe in structural terms how the organs are held in place. These two methods are the suspensory axes and DeLancey's Biomechanical levels. Both are illustrative and can be helpful when assessing or repairing compromised anatomy.

The primary suspensory axis is located in the posterior vagina and consists of a continuum of connective tissue between the vaginal introitus and the posterior bony pelvis.[24] The named anatomic structures are listed in Table 1.4.

An equally important anterior axis also exists. Its components are listed in the accompanying table. The anterior axis connects with the primary posterior axis through the pericervical ring. Reconnecting both of the axes within the interspinous diameter is exceptionally important in the eventual integrity of prolapse surgery. If the cervix has been removed, a cervical defect is present in the anterior axis, and if surgical measures are not taken to compensate for this defect, an inherent weakness is present. For that reason, apical suspension of the pubocervical septum is one of the key maneuvers in a prolapse repair. Notice that the pubocervical septum is shorter than the rectovaginal septum by a distance equal to the diameter of the cervix. Failure to recognize this fact can doom a repair to failure or unnecessarily shorten the vaginal depth (Table 1.5). Pelvic reconstructive surgery that does not accomplish a complete reestablishment of these axes will be more likely to fail. Note that the central area of reconnection should be the interspinous diameter. Pelvic reconstructive surgeons should be comfortable with the necessary dissection to accomplish this primary goal of pelvic reconstructive surgery.

DeLancey's biomechanical levels validate the concept of the suspensory axes.[21] They are well known and should be studied and conceptually mastered by all pelvic reconstructive surgeons (Table 1.6). Level I suspension is primarily dependent on the paired uterosacral ligaments with some structural contribution from the paired cardinal ligaments. Level II lateral attachment of the anterior and posterior septa is to the arcus tendineus fascia pelvis. In Level III, the vaginal

Table 1.4 Posterior suspensory axis of the uterovaginal complex

Perineal body
Rectovaginal septum
Pericervical ring
Uterosacral ligaments (paired)
Presacral periosteum of S2, 3, and 4

Table 1.5 Anterior suspensory axis of the uterovaginal complex

Urogenital diaphragm
Pubocervical septum
Pericervical ring

Table 1.6 DeLancey's biomechanical levels of uterovaginal support

Level I: Suspension
Level II: Lateral attachment
Level III: Fusion

fasciae fuse with relatively immobile structures anteriorly and posteriorly. Anterior fusion is to the urogential diaphragm. Posterior fusion is to the perineal body. Several conceptual similarities are shared between the concepts of the suspensory axes and the biomechanical levels. A good knowledge of these complimentary notions can assist prolapse surgeons plan procedures that ensure continuity of connective tissue between the vaginal introitus and the sacrum. In both systems, the central structure in both systems is the pericervical ring and its attachments. The endopelvic fascial structures form an integrated structure that allows central pelvic organs to be anatomically stable and functionally intact. Surgical restoration of the normal connective attachments within the interspinous diameter is the primary goal of the prolapse surgeon.

Conclusion

A thorough understanding of pelvic anatomy provides invaluable knowledge to the pelvic surgeon; however, structural anatomy is not sufficient. In addition to anatomy, normal form and function depend on an understanding of the biodynamics of pelvic structures in the normal state, the abnormal state, and during the process of parturition. Integrative knowledge of all of these concepts will allow surgeons to properly design and perform operations for pelvic reconstructive surgery.

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New Directions in Restoration of Pelvic Structure and Function

2

Peter E. Petros and Bernhard Liedl

The fundamental theme of this chapter is that structure and function are intimately related. *Abnormal symptoms and prolapse are caused by connective tissue laxity in the vagina or its suspensory ligaments – Integral Theory*[1] (Fig. 2.1). Other than pelvic pain, in some way, all the symptoms concern closure (continence) or opening (emptying) by the muscle forces (arrows). Tissue tension is critical for each of these functions. It follows that, in order to restore function, the surgical technique used must also restore tissue tension. A new tensioned sling technique which fulfills these criteria is presented later in this chapter. The tensioned sling works like the tensioned wires of a suspension bridge. It addresses both prolapse and abnormal symptoms, and has been successfully applied in >2,000 cases since November 2003 for patients with both stress incontinence and major prolapse.

There are three zones and nine potential sites of connective tissue damage in the female pelvis (Fig. 2.1). Correct diagnosis of which ligament(s) is damaged is critical, so as to guide accurate repair of such ligament(s).

Restore the structure, and you will correct the function.

Dynamic Anatomy

Organs are suspended by ligaments. Pelvic muscles (arrows, Fig. 2.1) stretch the organs against the ligaments to give them shape and support. By a sequence of coordinated contraction and relaxation, the organs are closed (continence) or are opened out actively (emptying). Lax ligamentous insertion points therefore may cause not only prolapse, but also symptoms of incontinence and abnormal emptying (Fig. 2.1). The Integral System of diagnosis and surgery is based on a three zone classification, containing nine connective tissue structures (Fig. 2.1).

Pathogenesis of Prolapse and Abnormal Symptoms

Abnormal symptoms and prolapse are caused by connective tissue laxity in the vagina or its suspensory ligaments – Integral Theory.[1]

The Causes of Damaged Connective Tissue

Childbirth, age, and congenital collagen defects are major causes of uterovaginal prolapse, bladder, and bowel dysfunction.

Structural Effects of Damaged Connective Tissue

The circles in Fig. 2.2 represent the baby's head descending down the vagina, stretching the connective tissue supporting structures (ligaments) laterally, thereby causing laxity. Lateral displacement of ligaments and fascia may cause the bladder, uterus, and rectum to herniate through the space to present as cystocoele, uterine prolapse, and rectocoele. The same ligamentous laxity may cause abnormal urinary and bowel symptoms (see Figs. 2.1, 2.3–2.5).

Minor Damage, Major Symptoms: The Trampoline Analogy

The three muscle forces tension the vaginal (trampoline) membrane against the suspensory ligaments (Fig. 2.6) (springs). Like a trampoline, laxity in even one ligament may prevent the vaginal membrane from being tensioned sufficiently to support the stretch receptors (N), and prevent them from activating the micturition reflex at a low bladder volume. The patient perceives this as frequency, urgency, and

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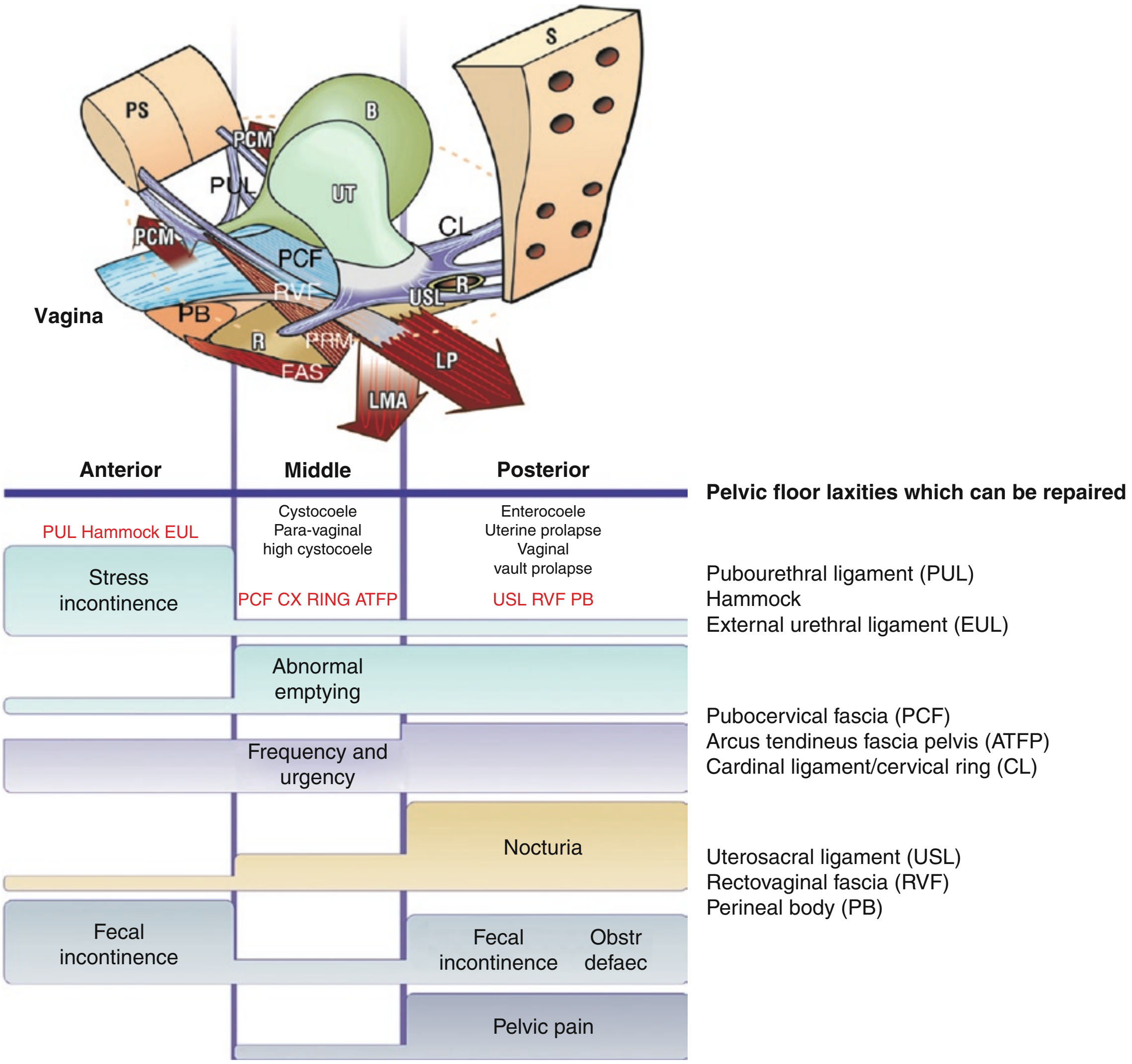
S
PS
B
PCM
PUL
UT
CL
PCM
PCF
RVF
USL
R
Vagina
PB
R
PRM
FAS
LP
LMA
Anterior
Middle
Posterior
Pelvic floor laxities which can be repaired
PUL Hammock EUL
Cystocoele
Para-vaginal
high cystocoele
Enterocoele
Uterine prolapse
Vaginal
vault prolapse
Stress
incontinence
PCF CX RING ATFP
USL RVF PB
Pubourethral ligament (PUL)
Hammock
External urethral ligament (EUL)
Abnormal
emptying
Pubocervical fascia (PCF)
Arcus tendineus fascia pelvis (ATFP)
Cardinal ligament/cervical ring (CL)
Frequency and
urgency
Nocturia
Uterosacral ligament (USL)
Rectovaginal fascia (RVF)
Perineal body (PB)
Fecal
incontinence
Fecal
incontinence
Obstr
defaec
Pelvic pain

Fig. 2.1 The pictorial diagnostic algorithm. A summary guide to causation and management of pelvic floor conditions. The area of the symptom rectangles indicates the estimated frequency of symptom causation occurring in each zone. The main connective tissue structures causing symptoms and prolapse in each zone are indicated in red capital letters. There is no correlation between degree of prolapse and symptom severity

nocturia. The cause may be ligamentous damage in any of the three zones. This statement can be directly tested by examining a patient with a full bladder. Digital pressure ("simulated operation") at midurethra controls stress incontinence and often urgency. Gentle digital support anterior to cervix or in the posterior fornix may also control urge symptoms.

Diagnosis

The pictorial diagnostic algorithm (Fig. 2.1) is the key to diagnosis.[2] It relates specific symptoms to damaged ligaments in each zone. Accurate assessment of the zone of damage by examination is critical. Often, the final diagnosis can only be made in the operating room.

Vaginal Examination

Each zone is examined, in turn, for damage and the results are recorded.

Anterior Zone Examination

The anterior zone extends from the external urethral meatus to bladder neck. Three structures are tested: the external urethral ligament (EUL), the pubourethral ligament (PUL), and the vaginal hammock. A "pouting" (open) external urethral meatus generally signals laxity in the EULs, especially if associated with eversion of the urethral mucosa (Fig. 2.7). The test for a damaged PUL

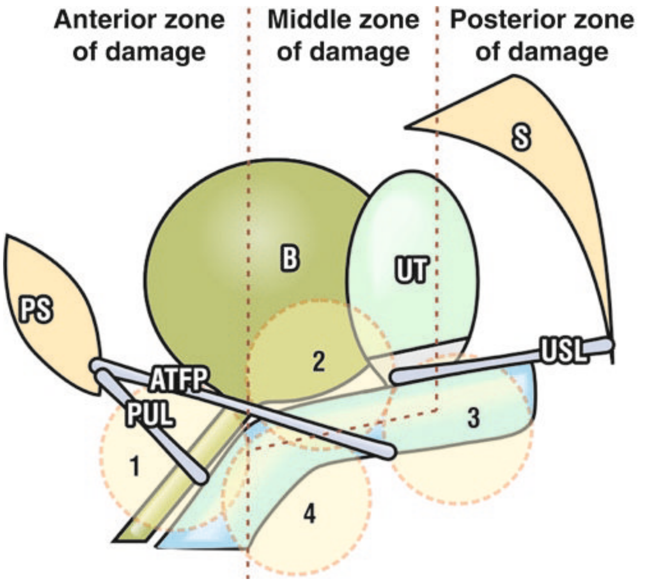

Fig. 2.2 Schematic representation of zones and structures of connective tissue damage at childbirth. (1) *PUL* pubourethral ligament (stress incontinence), (2) *ATFP* arcus tendineus fascia pelvis and pubocervical fascia (cystocoele), (3) *USL* uterosacral ligament (uterine prolapse), (4) Perineal body/rectovaginal fascia (rectocoele)

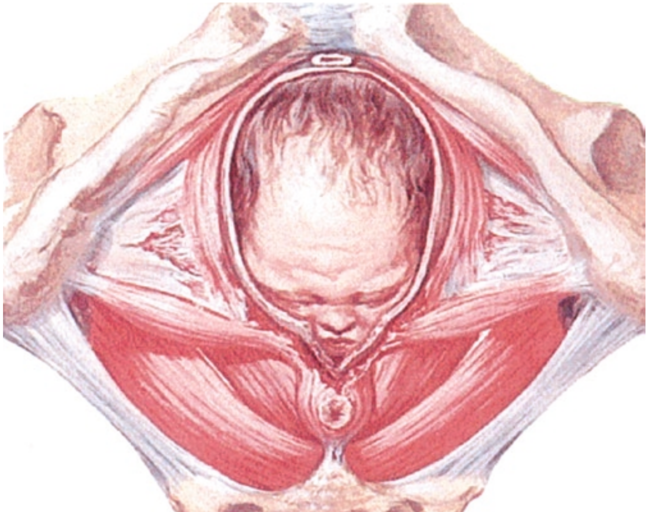

Fig. 2.3 Childbirth. Forcible lateral displacement of hiatal and perineal structures. The A-P diameter of the pelvis is 12–13 cm. A flexed head measures 9.4 cm, and a deflexed head 11.2 cm. The margin for prevention of damage is low (After Santoro)

involves two essential stages. The first is for the patient to demonstrate urine loss in the supine position on coughing. Then, a finger or a hemostat is placed at midurethra on one side and the cough is repeated. Control of urine loss signifies a weak PUL.

A lax hammock (see Fig. 2.7) is evident on inspection, but it can also be tested by the "pinch" test; taking a unilateral fold of the hammock with a hemostat. Diminution of urine loss during this test demonstrates the importance of an adequately tight hammock for urethral closure. These maneuvers are an essential part of the vaginal examination (Fig. 2.8).

Middle Zone Examination

The middle zone extends from bladder neck to the anterior lip of the cervix or hysterectomy scar. It has three connective tissue defects, central, lateral ("paravaginal"), and cardinal ligament/anterior cervical ring defect ("high cystocoele," "transverse defect"). A central defect typically is shiny, and "blows out" on straining. A central cystocoele can be differentiated from a paravaginal defect by placing ring forceps in the lateral sulci to support the ATFP and asking the patient to strain. Often, however, a patient has both central and lateral defects. The cardinal ligaments insert anteriorly into the cervical ring. Tearing of this insertion may dislocate the pubocervical fascia, creating a characteristic lateral extension of the bladder fascia around the cervix (Fig 2.9, arrows). This is known as a "high cystocoele" or "transverse defect" and it is often accompanied by a retroverted or prolapsed uterus (Figs. 2.10 and 2.11).

Posterior Zone Examination

The posterior zone extends from the cervix/hysterectomy scar to the perineal body. Evidence of a bulge at the apex, vaginal wall, or perineal body should be looked for during straining. Small degrees of prolapse in the apex of the vagina are easily missed. Therefore, when examining in the supine position, always support the lateral sulci of the anterior vaginal wall with ring forceps and ask the patient to strain when examining the posterior zone. Alternatively, examination in the left lateral position may be helpful. The posterior vaginal wall is tested for defects in the rectovaginal fascia (rectocoele) by asking the patient to strain, and also by digital rectal examination. The perineal body and external anal sphincter are tested by digital examination. Major posterior zone defects are frequently accompanied by other defects. For example, the patient (Fig. 2.12) most likely has a cardinal ligament/cervical ring defect, a central cystocoele, lax and separated uterosacral ligaments with an enterocoele, and probably lax rectovaginal fascia (Fig. 2.13).

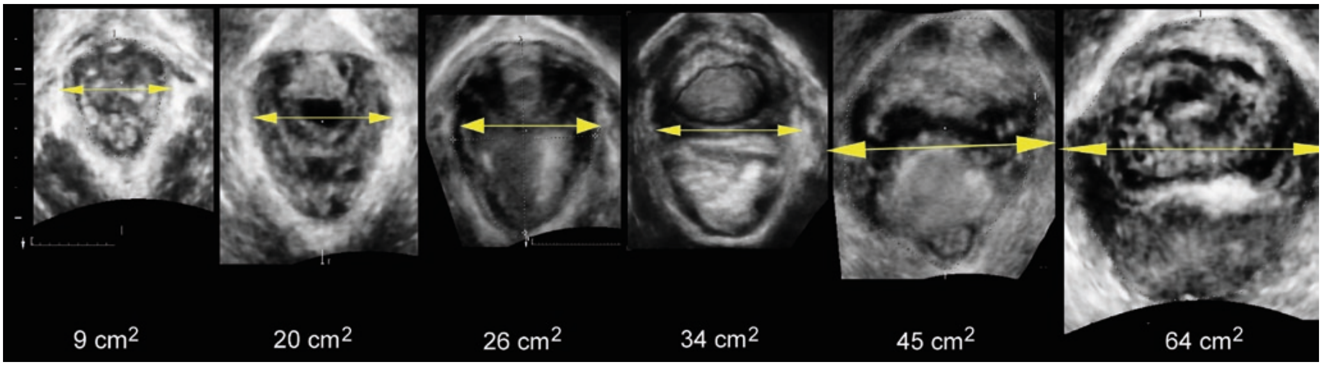

Fig. 2.4 3D ultrasound demonstrates a dramatic widening of the levator hiatus "ballooning," in a patient during straining (Valsalva), from 9 cm^2 at rest, to 64 cm^2 (After Dietz HP. With permission). The *arrows* define the hiatal space between the pubovesical muscles. This figure is consistent with the causation proposed in Figs. 2.2 and 2.3: connective tissue damage of the ligaments and fascia binding the hiatal structures causes lateral displacement, laxity, and herniation of these structures

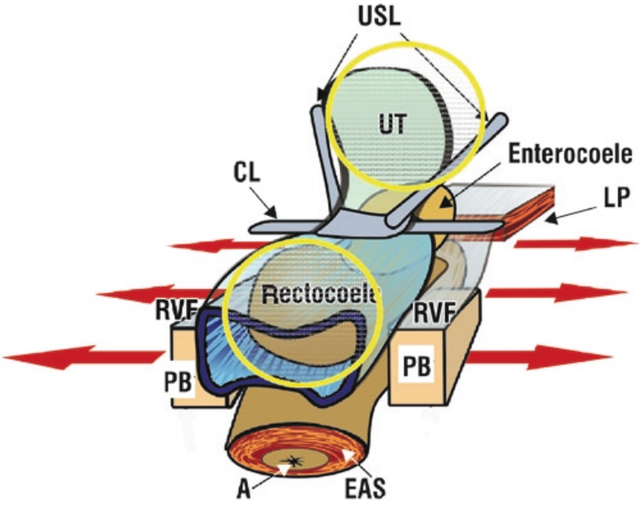

Fig. 2.5 Childbirth. Forcible lateral displacement of uterosacral ligaments (*USL*), perineal body (*PB*) and rectovaginal fascia (*RVF*) by the fetal head (*circles*) causing connective tissue laxity, and protrusion of enterocoele and rectocoele

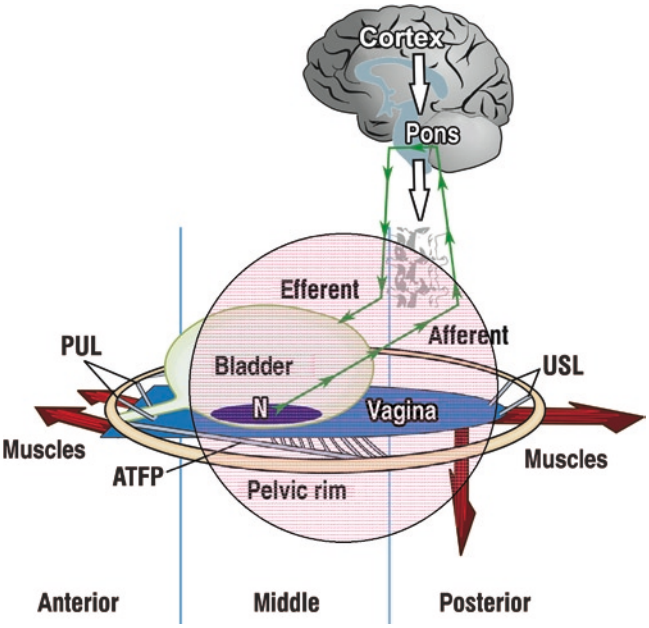

Fig. 2.6 Schematic representation of a fetal head pressing into the pelvic brim, against the vagina and its suspensory ligaments, uterosacral (*USL*), pubourethral (*PUL*), and arcus tendineus fascia pelvis (*ATFP*). Even minor damage to the ligaments may cause urgency, as this symptom is neurologically determined

Surgical Repair of Connective Tissue Structures

Reconstructive pelvic floor surgery according to the Integral Theory System differs from conventional surgery.[3]

1. It has a symptom-based emphasis (the pictorial diagnostic algorithm), which expands the surgical indicators from major prolapse to include cases with major symptoms and only minimal prolapse. The same operations apply for symptoms and prolapse.
2. Special instruments insert polypropylene tapes to reinforce damaged ligaments in three zones of the vagina.
3. It is based on specific surgical principles which minimize risk, pain, and discomfort to the patient.
   To minimize pain
   - Avoid tension when suturing the vagina
   - Avoid vaginal excision
   - Avoid surgery to the perineal skin
   To avoid urinary retention
   - Avoid tightness in bladder neck area of vagina
   - Avoid indentation of the urethra with a midurethral sling
4. The uterus needs to be conserved wherever possible. It is the central anchoring point for the posterior ligaments, the rectovaginal fascia, and the pubocervical fascia. The descending branch of the uterine artery is a major blood supply for these structures, and should be conserved where possible even if subtotal hysterectomy is performed.

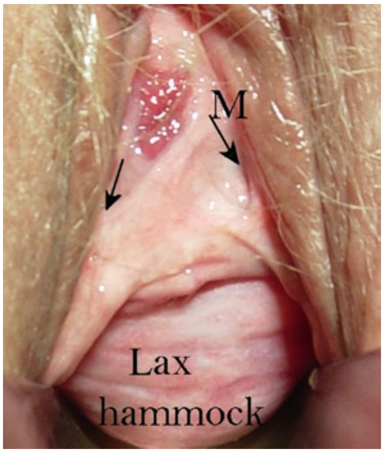

Fig. 2.7 Lax external urethral ligament (*EUL*) and hammock. The urethral meatus (M) is lax, and the urethral mucosa is everted. The lateral EUL supports are seen "drooping" downward (*arrows*). The hammock is lax and angulated downward

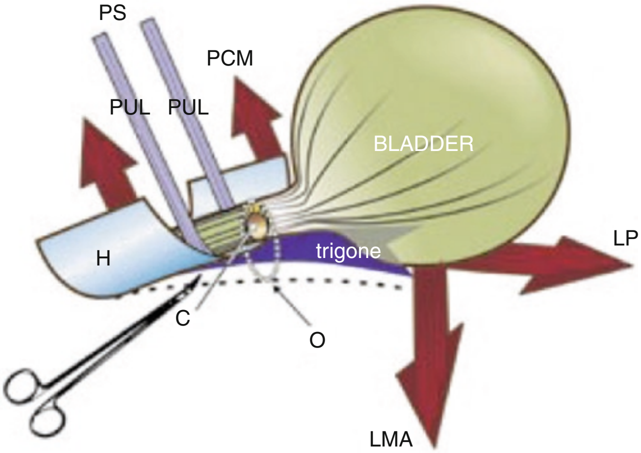

Fig. 2.8 Testing for a lax pubourethral ligament.[2] Unilateral anchoring at midurethra is the only method possible for diagnosing a damaged pubourethral ligament (*PUL*). Cessation of urine loss on coughing confirms a lax PUL. Midurethral anchoring restores the closure forces, which narrow the urethra from "O" (stress incontinence) to "C" (continence) during coughing, and it also restores the geometry from a funneled to a normal outlet. Taking a fold of vagina "H" ("pinch" test) generally also decreases urine loss

"Tension-free Slings" (with or Without Attached Mesh)

These techniques are designed to reinforce damaged ligaments and fascia, and are well covered by other contributors. This chapter concerns "New Directions," in particular, tensioned minislings, as applied for prolapse and abnormal symptoms.

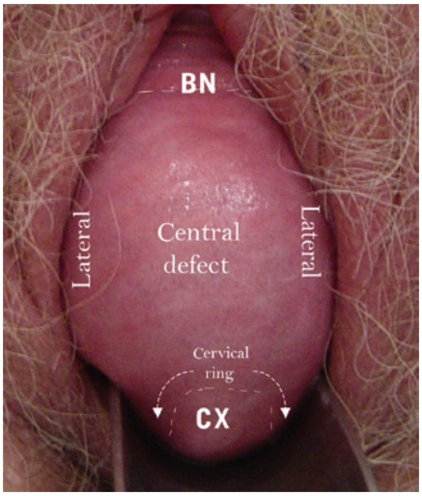

Fig. 2.9 Prolapse, third degree, of bladder and uterus. A large central defect extends laterally. Prolapse of bladder around cervix (CX) (*curved arrows*) is characteristic of Cardinal ligament/cervical ring defect. *BN* bladder neck

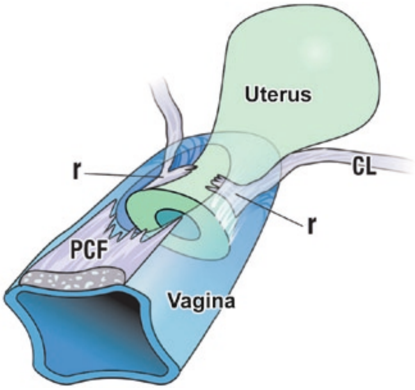

Fig. 2.10 A ruptured cervical ring "r" may cause dislocation of PCF (cystocoele). It may loosen the cardinal ligament attachments "CL," so that the uterus may retrovert and even prolapse

Tensioned Minislings: A Physiological Alternative for Prolapse Repair

"Minislings" mostly avoid the major vascular and nerve complications reported with the retropubic, transobturator, and perineal slings. The tensioned minisling (Fig. 2.14) applies the engineering principles of a suspension bridge – the suspensory wires (ligaments) (Fig. 2.14) hold up the suspension bridge (pelvic organs). Lax ligaments cannot support the organs, resulting in prolapse. Unlike large mesh sheets, there is no limitation to backward extension of the organs, because the tapes are transversely sited (Fig. 2.14). There is no invasion of the rectovaginal and vesicovaginal spaces, so scarring, adhesions, and dyspareunia are largely avoided.

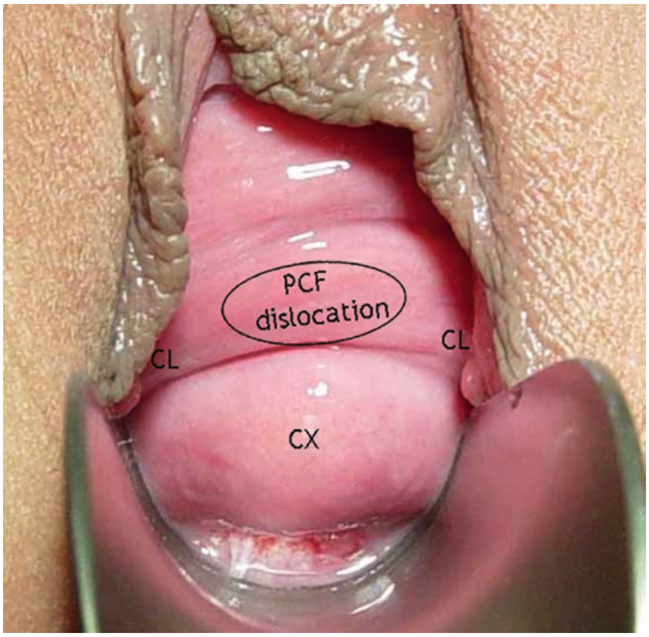

Fig. 2.11 Differentiation between central/lateral cystocoele and high cystocoele (cardinal ligament/cervical ring defect) is confirmed if the cystocoele disappears when laterally placed Allis forceps are approximated. Persistence of a bulge indicates the lesion is caused by a rupture/stretching of the pubocervical fascia (central/lateral defect)

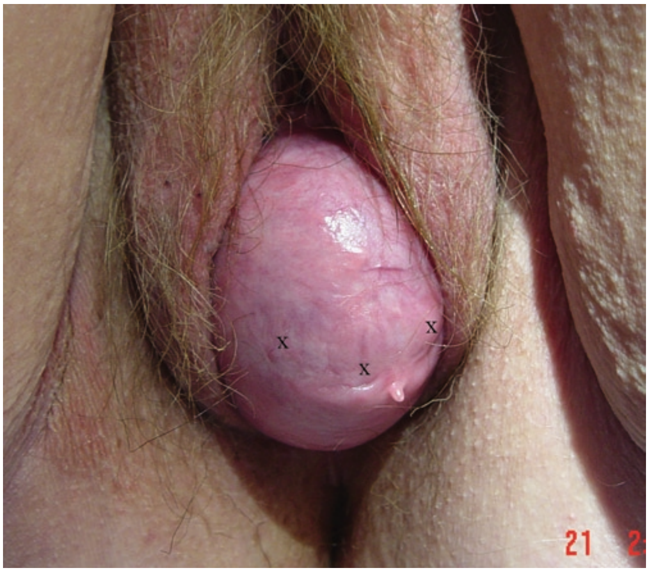

Fig. 2.12 Everting fourth degree vault prolapse. X denotes the line of the hysterectomy scar

The “cathedral ceiling” analogy visually explains how tapes (joists) can provide support to a much weaker structures such as damaged vaginal fascia (Fig. 2.15).

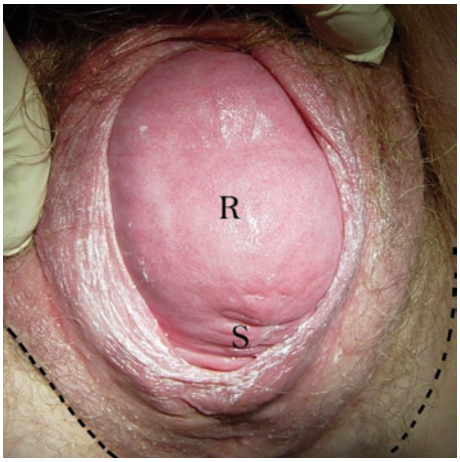

Fig. 2.13 Large rectocoele (R), and deficient perineal body (*broken lines*) revealed by rectal examination. Note scar “S” from previous surgery for rectocoele

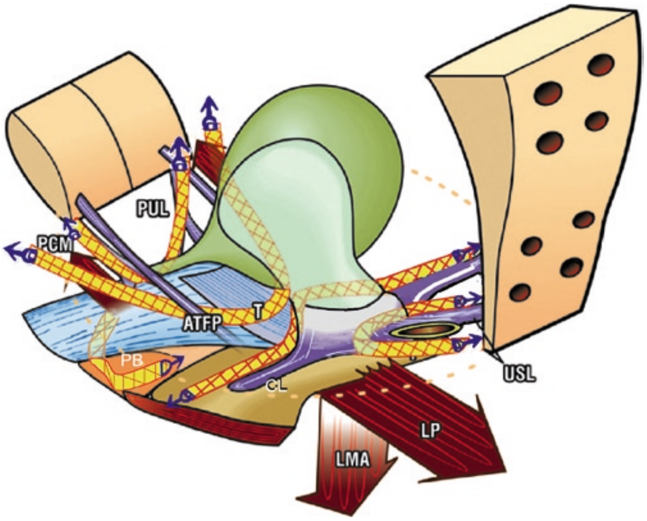

Fig. 2.14 Tensioned Polypropylene tapes “T” bring the laterally displaced ligaments and fascia toward the midline. This tightens the suspensory ligaments like the wires of a suspension bridge, and the tapes create artificial neoligaments to bind the connective tissue structures together during straining (see Fig. 2.4): pubourethral (*PUL*), uterosacral (*USL*), cardinal (*CL*), arcus tendineus fascia pelvis (*ATFP*), and also, perineal body (*PB*)

Symptom Cure

Connective tissue must be tensioned to restore muscle function (Fig. 2.6) because a muscle requires a firm insertion point to function optimally. The descriptions below are confined to the TFS (Tissue Fixation System) minisling, as that is the only tensioned sling available today. Short-term results in patients with multiple symptoms and symptom improvement

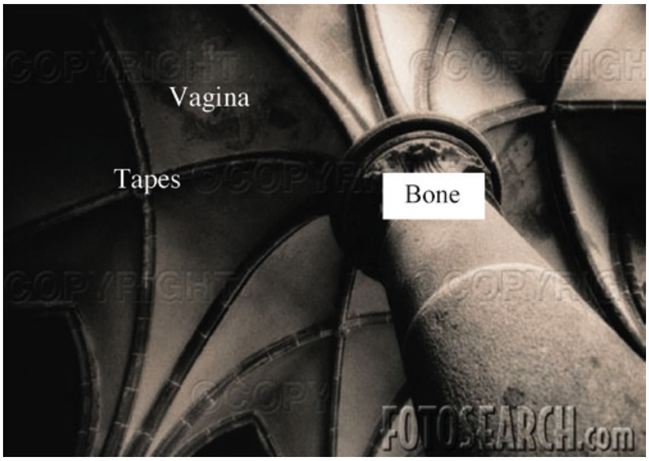

Fig. 2.15 The cathedral ceiling structural analogy – a new direction for prolapse *repair.* Direct reinforcement of the ligaments, as in Fig. 2.14, provides sufficient strength for prolapse repair, without the requirement for large mesh

were as follows: SI (89%) fecal incontinence ($n = 33$), 88%, stress incontinence ($n = 43$), 89%, urgency and nocturia ($n = 50$), 80%.[4]

Tensioned Midurethral TFS Minisling: Repair of the Pubourethral Ligament

Indications

Stress incontinence (SI) or mixed incontinence. The dissection is almost identical to a “tension-free” tape sling – a midline incision, dissection of urethra from vagina, penetration of the perineal membrane (urogenital diaphragm). The applicator is placed into the dissected space. The TFS anchor is released and the tape tightened over an18G Foley catheter until it touches the urethra without indenting it. The free ends are trimmed. The vaginal hammock fascia and the external ligamentous attachment of the external urethral meatus are then tightened with 2–0 Vicryl sutures. No cystoscopy is required. The cure rate at 3 years is equivalent to “tension-free” midurethral tape operations[5] (Fig. 2.16).

Tensioned Pre-pubic TFS Minisling: Repair of the External Urethral Ligament

Indications

Continued urine leakage after cure of stress incontinence with a midurethral sling

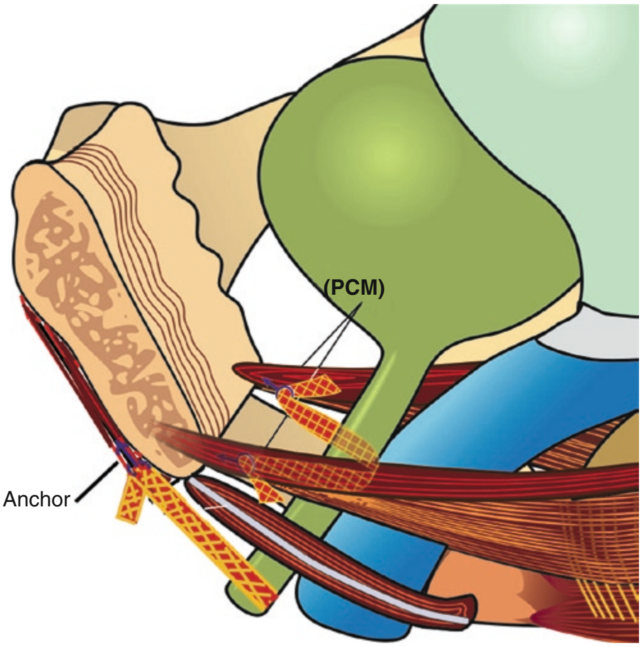

Fig. 2.16 Anatomical position of the Tissue Fixation System (*TFS*) anchor. The midurethral tape is anchored into the inferior surface of the pelvic floor muscles. The prepubic (external ligament) TFS tape is positioned between the muscle layer and tissue covering the anterior surface of the pubic bone

The patient complains of leakage on sudden movement, often associated with a feeling of a “bubble” escaping. There is usually no SI. Measured leakage may be large, but is reduced by 50–70% by insertion of a menstrual tampon. The operation is identical to a midurethral sling, except that that channel is made anteriorly, between the anterior surface of the pubic bone and the muscle layers.

Tensioned TFS Mini “U” Sling Repairs Central and Lateral Pubocervical Fascia and ATFP

Indications

Cystocoele caused by a central/lateral (paravaginal) defect.

The surgical principle underpinning this operation is to mimic the ATFP and to provide a transverse neofascial “beam” to reinforce the damaged central pubocervical fascial defect. In patients with an intact uterus and no previous surgery, the dissection can be made via a transverse 2.5–3 cm incision at the vesical fold. The bladder is dissected off the vaginal wall and cervix. Under tension, a channel is made below the pubic ramus, extending onto the medial aspect of the obturator fossa, in the position of the ATFP insertion (Fig. 2.17). The applicator is inserted, the anchor released and the tape tightened until a resistance is felt. The vagina is sutured without tissue

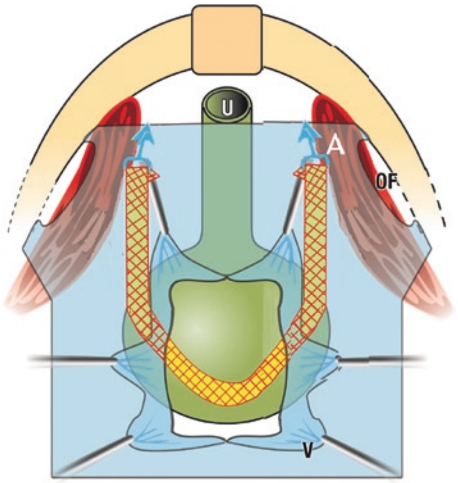

Fig. 2.17 "U" sling. View into the anterior vaginal wall. Vagina (*V*) is dissected off the bladder wall, and stretched laterally. The TFS tape is anchored (*A*) medial to the obturator fossa (*OF*) muscles, toward the arcus tendineus fascia pelvis (*ATFP*)

excision. In patients with previous hysterectomy or previous vaginal repair, an inverted T incision is made to ensure adequate dissection and reduce the risk of bladder perforation.

Cervical Ring "Transverse" Defect (High Cystocoele) Repair

Indications

Cystocoele caused by an anterior cervical ring/cardinal ligament defect, especially if associated with urgency and abnormal emptying symptoms.

This is a common lesion, especially after hysterectomy, which necessarily dislocates the attached cervical ring and attached fascia. A 2.5–3 cm horizontal incision is made in the vesical fold 1cm above the hysterectomy scar, or above the cervix. The bladder is dissected clear of the vagina and cervix. A channel is made along the cardinal ligament to just beyond the lateral sulcus. The dissection plane is about 2 cm above the ischial spine. The TFS applicator is inserted, the anchors released, and tape tightened until a resistance is felt. A high initial cure rate at 9 months has been achieved for TFS cystocoele repair[5] (Fig. 2.18).

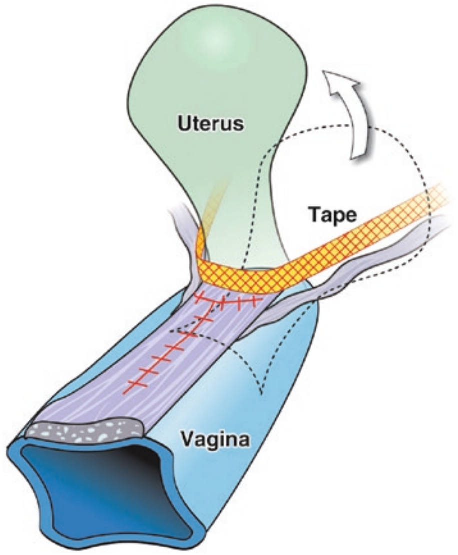

Fig. 2.18 Cervical ring/cardinal ligament repair, sagittal view. The tape is placed along the anterior lip of cervix and extends along the cardinal ligament. On tightening, the cervix is pulled back, and the uterus anteverts

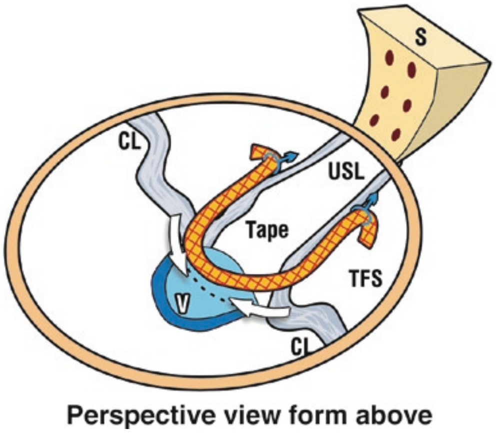

Fig. 2.19 Posterior TFS. Perspective: View from above. The tape is placed along the exact position of the uterosacral ligament (*USL*). The arrows indicate how the remnants of USL are approximated during tightening, closing the enterocoele

The Posterior TFS Sling

Indications

Uterine/apical prolapse, enterocoele: In patients with significant "Posterior Fornix Syndrome" symptoms (nocturia, pelvic pain, urgency, abnormal bladder emptying, Fig. 2.1), this operation is performed even with minimal prolapse. The results at 3 years (unpublished data) are equivalent to more invasive procedures.

The posterior TFS sling is similar to the McCall operation insofar as it anchors the apical fascia into the uterosacral ligaments (USL). A full thickness, 2.5–3 cm transverse or longitudinal incision is made in the vaginal apex. The uterosacral ligaments (USL) or their remnants are identified and grasped with Allis forceps. If an enterocoele is present it is reduced. Fine dissecting scissors create a 4–5 cm space just lateral to the USLs for the instrument. The anchors are ejected, and the tape tightened. Tightening the tape approximates the uterosacral ligaments and closes the enterocoele (Fig. 2.19).

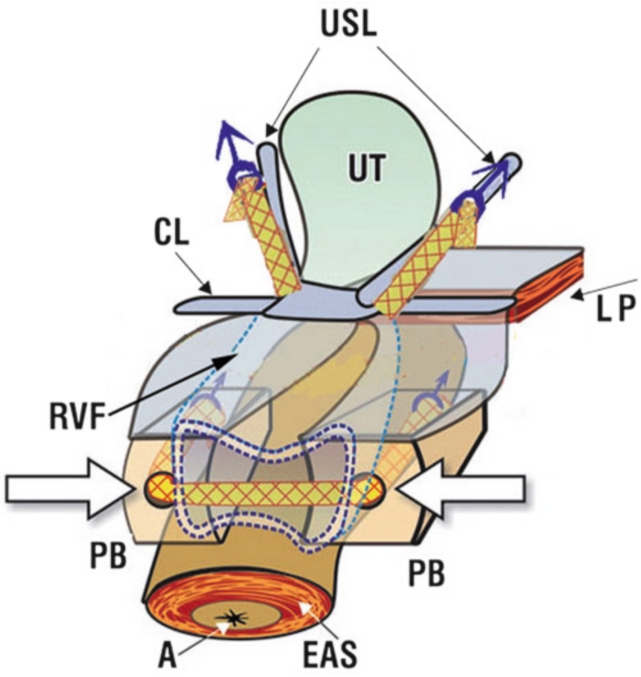

Fig. 2.20 Approximation of laterally displaced perineal body and RVF. Inferiorly, the TFS strongly approximates the laterally displaced perineal body (*PB*), and with it, rectovaginal fascia (*RVF*). Superiorly, the posterior sling approximates the laterally displaced uteroscral ligaments and attached fascia, at the same time closing the enterocoele

Posterior TFS Sling at the Time of Vaginal Hysterectomy

Vaginal vault prolapse is a major long-term complication of hysterectomy. Posterior TFS sling during vaginal hysterectomy is simple, takes only a few minutes to perform, yet provides strong vaginal vault support at 12 month review (Petros and Richardson, unpublished data).

Perineal Body TFS Sling

A stretched perineal body is the condition where the perineal body (PB) has been stretched thinly across the lower part of the anus. During surgical reconstruction a transverse incision just inside the muco-cutaneous junction vastly facilitates access to the laterally placed intact parts of the perineal body. Using dissecting scissors, and controlled by rectal examination, a channel is made vertically into the body of each perineal body to just beyond the insertion of deep transverses perinea to the inferior pubic ramus (Fig. 2.20). The tape is "set" and tightened. This brings each perineal body toward the midline and adequately closes a low and mid-rectocoele.

Limitations of Minisling Surgery

Whereas a midurethral sling operation is significantly simpler than the retropubic or transobturator method, a good working knowledge of the site of the pelvic ligaments is required for prolapse surgery. Accurate anchor placement in the position of damaged ligaments is required for tensioned slings to work. Organ damage to date has been minimal.

Potential Longer-Term Complications of Minisling Surgery

The main complications are erosion, and change in the structural balance of the three zones. The more significant complication is the development of de novo prolapse and symptoms in other compartments weeks or months after surgery, because structural reinforcement in one zone may divert the pelvic muscle forces to other subclinically weakened zones. Failure to cure may be due to wrong diagnosis, decompensation of other connective tissue structures caused by the intervention itself, or surgical failure of the operation itself. Repetition of the preoperative protocol, diagnosis of the zone and structure(s) (Fig. 2.1) that have been damaged, cough and 24-h pad tests to assess seriousness of the problem, are the key elements in the decision tree for management.

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3

Hernia Principles: What General Surgeons Can Teach Us About Prolapse Repair

Richard I. Reid

Unlike knee or ankle ligaments, *pelvic connective tissue is NOT structurally suited to chronic load bearing.*[1] Hence, Nature relies upon a complex inter-relationship between the pelvic floor muscles and the connective tissues.

- The pelvic floor muscles have two main roles: they narrow the gap through which the urethra, vagina and anus exit the abdomen; and they also form a dynamic backstop to actively oppose intra-abdominal pressure. Hence, the pelvic floor muscles absorb most of the expulsive load on the pelvic organs, and it is very difficult for the body to compensate any muscle damage.[2]
- Pelvic connective tissue is also important, but in a less direct way. The primary suspensory role of the fascia is to attach the organs to the pelvic skeleton, thus stabilizing them over the center of the muscular plate.

Traditional gynecologic strategies for prolapse repair have depended unduly upon endopelvic fascial strength. Hence, experience accrued by herniologists in averting healing failure due to collagen weakness has useful lessons for the pelvic reconstructive surgeon.

Before exploring the "hernia hypothesis" in more detail, we need to resolve the common confusion between "fascia" and "aponeurosis." Surgeons tend to use these two terms interchangeably, but such usage is not anatomically correct.[3]

- The term "aponeurosis" means a flat tendinous sheet connecting a striated muscle to a fixed point on the bony skeleton. Collagen bundles within an aponeurosis are oriented into parallel arrays, coincident with the lines of force – thus conferring extreme internal strength. By serving as a flat expanded tendon, an aponeurosis transitions between the muscle fibers and their point of bony insertion, partly safeguarding these vulnerable areas from trauma. Relevant examples of an aponeurosis would be the "rectus sheath" (the aponeurosis covering the rectus abdominus muscles in the abdominal wall), the "transversalis fascia" (the aponeurotic termination of the deepest of the three abdominal strap muscles), the "obturator fascia" (the aponeurosis – not fascia, as the name implies – covering obturator internus muscle on the inside of the pelvic bones), and the "perineal membrane" (the aponeurosis covering the small muscles of the urogenital diaphragm).
- The term "fascia" simply refers to any connective tissue that has condensed into a layer that can be seen with the naked eye. Fascia is strong, but only moderately so. Collagen bundles have a random (rather than linear) organization. The real function of fascia in the body is to serve as a fibro-fatty investment covering the underlying muscles and their aponeuroses. This fibro-fatty investment provides body contour, insulation, and acts as a conduit for surface blood and lymphatic vessels.

The Hernia Hypothesis

Hernia is the protrusion of an internal organ (usually small intestine) through a weakness in the abdominal wall. The pathogenesis of hernia has two components.[4]

- *A mechanical event:* Namely, a "site-specific" defect in the aponeurotic layers investing the peritoneal cavity. Such weakness can arise as a congenital weakness at the internal ring[5] or a traumatic/post-incisional break in the transversalis fascia.[6] Any protruding tongue of peritoneum generally remains subclinical for years; however, progression to symptomatic hernia becomes likely if abdominal wall strength can no longer contain the intra-abdominal forces generated during Valsalva straining or at loading of the torso during heavy exertion.[7,8] Hernia formation is also favored by any genetic compromise of connective tissue quality.
- *A metabolic event:* Namely, primary (genetic) or secondary (acquired) degenerative weakness in the aponeurotic tissue adjacent to the initial defect.[9-11] Such degeneration in collagen quality inevitably occurs when bones, ligaments or tendons are not involved in continuous remodeling in response to body forces.[12]

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Likewise, *prolapse* is the protrusion of an organ (uterus, bladder or bowel) through the vaginal fibromuscularis, usually at a site of childbirth injury. It is also has mechanical and metabolic components.

- The *mechanical event* is a group of "site-specific" tears in the endopelvic fascia, most commonly arising through childbirth injury.[13] The likelihood of mechanical failure is increased by any concomitant pelvic myopathy or neuropathy. Progression from subclinical anatomic laxity to symptomatic prolapse is greatly influenced by the operation of diverse secondary factors (Table 3.1).[14,15]
- The *metabolic event* is also collagen weakness, either inherited or acquired. Patients with inherited collagen disorders (like Ehlers Danlos or benign joint hypermobility syndromes) have a high incidence of prolapse; treatment is also more likely to fail.[16,17] However, biochemically normal women with chronic prolapse often develop an acquired metabolic collagen weakness,[18-20] because the mechanical forces that drive homeostasis are not properly transmitted within torn suspensory hammocks.[21,22]

The modern era of herniology began with Bassini's description of a "site-specific" repair of defective transversalis fascia on the floor of the inguinal canal in 1887.[23] Despite innumerable technical modifications over the succeeding century, long-term recurrence rates from tissue approximation repairs remained in the 15–33% range.[24-26] Likewise, in several regional[27,28] and national[29] surveys, recurrence rates for Mayo reduplicative repair of incisional hernia have remained around 25–54%.[30-34] In that these high failure rates are not attributable to overt technical errors, the possible role of connective tissue factors has received increasing attention.[35-65] Hereditary tissue weakness is known to predispose to both hernia and prolapse; there is also mounting evidence of acquired connective tissue weakness in genetically normal individuals, secondary to disrupted collagen homeostasis tissues in long-standing hernia and prolapse (Table 3.2).

The History of Hernia and Prolapse Surgery

Ancient Times

Hernia and prolapse were well described as long ago as 400 BC, notably by Hippocrates in ancient Greece and Celsus in ancient Rome. However, the pathogenesis was not understood, and nobody at that time envisaged an effective surgical cure for either problem. Physicians had nothing but ineffective medical treatments and occasional primitive operations for the next 2,000 years, from the time of Hippocrates to the beginning of Elizabeth I's reign. In this same era, women with prolapse were managed by being suspended upside down or by wearing a half pomegranate in the vagina as a pessary (Fig 3.1).

Table 3.1 Factors in the evolution of pelvic organ prolapse (Modified after Bump and Norton[14])

Predispose	Incite	Promote	Decompensate
Race (White > Asian > Black) Benign joint hypermobility syndrome Hereditary collagen weaknesses: • Ehlers Danlos' syndrome • Marfan's syndrome • Osteogenesis imperfecta Congenital myopathy or neuropathy (e.g., spina bifida variants)	Pregnancy Vaginal delivery (fascial tears, avulsive and denervating myopathy) High impact trauma to pelvic floor: • Parachute jumping • Motor vehicle accident • Fractured pelvis	Tobacco smoking Chronically raised intra-abdominal pressure • Pulmonary disease (chronic cough) • Constipation (chronic straining) • Recreational or occupational heavy lifting • Obesity Altered force vectors following prior pelvic reconstructive surgery. • Enterocoele promotion by pulling vaginal axis too far forward at prior Burch colposuspension. • Cystocele promotion by pulling vaginal axis too far backward at prior sacrospinous fixation.	Aging Andropause and menopause General debility and other catabolic syndromes Malnutrition syndromes: • Protein-caloric subnutrition (as evidenced by low serum albumen); • Vitamin C, A, B_6 deficiency (needed for collagen synthesis); • Vitamin B_1, B_2, zinc, and copper deficiency (needed for wound repair) Medication (corticosteroids, ?ACE inhibitors) Vaginal gaping, exposing residual pelvic supports to chronic load • Laceration of the perineal membrane/perineal body complex • Chronic divarication of levator ani muscles

Table 3.2 Evidence for the operation of analogous collagen disorders in hernia and prolapse

Parameter	Hernia	Prolapse
Main initiating factor	Weakness in the investing aponeurosis surrounding the celomic cavity. Decompensation from subclinical laxity to symptomatic hernia is more likely if abdominal muscle strength cannot contain the forces generated during Valsalva straining or torso loading at heavy lifting.	Avulsion of the uterosacral ligaments or mid-vaginal septae from the pericervical ring at vaginal delivery. Obstetric trauma almost always occurs in the plane of the ischial spines, usually during first stage of labor.[36] Progression from asymptomatic anatomic laxity to overt prolapse is influenced by a variety of secondary factors, including a decline in local connective tissue quality.
Higher incidence and recurrence in wgenetic collagen disorders	Higher incidence and recurrence rates in Ehlos Danlos and Marfan's syndromes.[9,37] Incisional hernia rate after laparotomy for abdominal aortic aneurysm (a marker of collagen weakness) was twice as high as with an equivalent midline incision for ilio-femoral bypass of an occluding thrombus.[12,38-43]	Higher incidence and recurrence rates in Ehlos Danlos, Marfan's, benign joint hypermobility syndromes and chronic corticosteroid use.[16,17,44]
Time curve of surgical recurrence	Cumulative 10-year recurrence rate in the Danish inguinal hernia registry forms an almost linear curve.[25,45] This is not the geometric pattern that would be seen if recurrence occurred solely from technical error at the initial surgery.	Life table analysis implicates both mechanical factors and collagen weakness as independent failure mechanisms.[12,46]
Role of tissue fatigue	In a retrospective, population-based cohort study of inguinal hernia from a Washington State hospital discharge database (1987–99), 5-year re-operation rate rose from 23.8% after a first failure, to 35.3% after a second, and 38.7% after a third recurrence. These differences would have been higher, but for the fact that synthetic mesh use almost doubled over this 12-year period, rising from 34.2% in 1987 to 65.5% in 1999. Controlling for age, sex, comorbidity index, year of the initial procedure and hospital descriptors, the principal hazard for operative failure proved to be the use or non-use of tissue augmentation material. A decision to perform a "suture-only" repair instead of a mesh hernioplasty increased higher recurrence rate by 24.1%.[27]	Five-year re-operation rate for sutured repair was reported as being 42% higher in recurrent prolapse, despite repeat surgery being done in a tertiary unit.[47]
Limitations of native tissue repair	In a multicenter RCT comparing "suture-only" and mesh hernioplasty in 200 incisional hernia patients, 10-year cumulative recurrence rate was twice as high if mesh had not been used (63% vs 32%).[48,49] There is also evidence that poor healing poses a significant limitation to the efficacy of tissue approximation repair in groin hernia. In a prospective Denmark-wide study, 5 year re-operation rates for the Lichtenstein inguinal hernia repair (a tension-free mesh onlay technique) were only one quarter that following the traditional Shouldice procedure (an open musculo-aponeurotic re-approximation, using sutures under tension).[45,50] A Cochrane analysis of 20 prosthetic hernioplasty trials came to similar conclusions.[51]	Use of tissue augmentation material delivered 23% improvement in 5-year durability in cystocele repair, relative to a mechanically analogous vaginal paravaginal repair. The bridging graft simplified the technical task of VPVR (reducing technical failure from 18.6% to 4.6%), and also rejuvenated adjacent connective tissue (reducing prolapse recurrence from 14.6% to 4.9%).[12]

(continued)

Table 3.2 (continued)

Parameter	Hernia	Prolapse
Biochemical evidence of diffusely disordered collagen metabolism	Biopsies from hernia patients show higher collagen type III: I ratios and abnormal fibroblast function. The abnormal type III: I ratio denotes a reduced proportion of high tensile strength (type I) collagen and an excess production of immature (type III) collagen.[10,52-56]	Biopsies from prolapse patients show reduced total collagen content and higher collagen type III: I ratios.[19,57,58] Such failures do not reflect tissue thinning in prolapse women – in fact, the vaginal muscularis layer in enterocoele has been shown to be thicker than normal.[59]
Possibility of disturbed local collagen homeostasis	Fascia and aponeurosis are metabolically active structures characterized by a dynamic equilibrium between stimulatory growth factors and lytic tissue collagenases (mainly matrix metalloproteinases 1, 2, 9, and 13).[52,60,61] This homeostatic balance is also partly regulated by the mechanical forces acting on the tissues,[60,62,63] and is thus disturbed by laceration of the adjacent investing fasciae. Disordered MMP activity has also been reported, but precise patterns are inconsistent.[10]	It is probable that endopelvic fascia in biochemically normal women can also acquire a metabolic collagen weakness, if day-to-day mechanical forces are not transmitted within a torn suspensory hammock.[60,64,65] Prolapse tissue biopsies have been shown to contain up to four times higher levels of lytic protease enzymes (as indicated by MMP activity).[18-20]
Disordered smooth muscle function	Not relevant	In addition to collagen abnormalities, there is a suggestion of disordered function of the smooth muscle component of the vaginal wall in prolapse. Boreham[57,58] reported a reduced proportion of physiological smooth muscle and an increased proportion of disorganized smooth muscle bundles, with decreased α-actin staining.

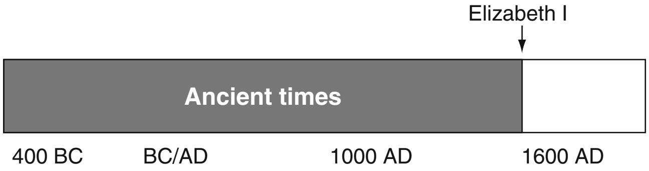

Fig. 3.1 Although both hernia and prolapse were well described by Hippocrates, there were no effective treatments and nothing much changed until the end of the dark ages

The Herniology Era

Interest in hernia treatments revived during the Renaissance of the sixteenth and seventeenth centuries, and some isolated (but notable) advances were made.[66]

- The first step on the road to modern hernia surgery was taken in 1559 by a Balkan surgeon called Kasper Stromagyi, who successfully treated a strangulated hernia by incising the skin, ligating the hernia sac at the external ring, and then sacrificing the testicle. The wound healed by secondary intension, and the patient survived. This was an astounding result for that era.
- One hundred and forty years later, a German surgeon called Purmann rescued a second strangulated hernia patient by a similar low ligation of the sac at the external ring. However, Purmann spared the testicle, rather than sacrificing it.
- These two insights led to sporadic attempts to manage hernia by scarifying the roof of the inguinal canal, typically by burning the aponeurosis of the external oblique with acid or hot cautery. As one would expect, results were absolutely miserable.
- The concept that a hernia bulge could be controlled by thickening the overlying fascia was refined in the mid-Victorian era, when Vinzenz von Czerny reinforced the roof of the inguinal canal with sutures. This strategy avoided having to incise the external oblique aponeurosis and enter the canal itself.

Thus was born the surgical technique of plication. This flourished among hernia surgeons for about 10 years, but was abandoned a decade later because of the 90% recurrence and 7% septic mortality rates. By comparison, the concept of plicating cystocele or rectocele was embraced by J. Marion Sims just after the American Civil War; however, there was very little actual treatment of prolapse until after World War I. It is disappointing that *gynecologists* adopted plication of prolapse long after *general surgeons* had abandoned the technique as being palliative (rather than curative) (Fig 3.2). It is even more disappointing that many gynecologists have kept right on plicating into the twenty-first century.

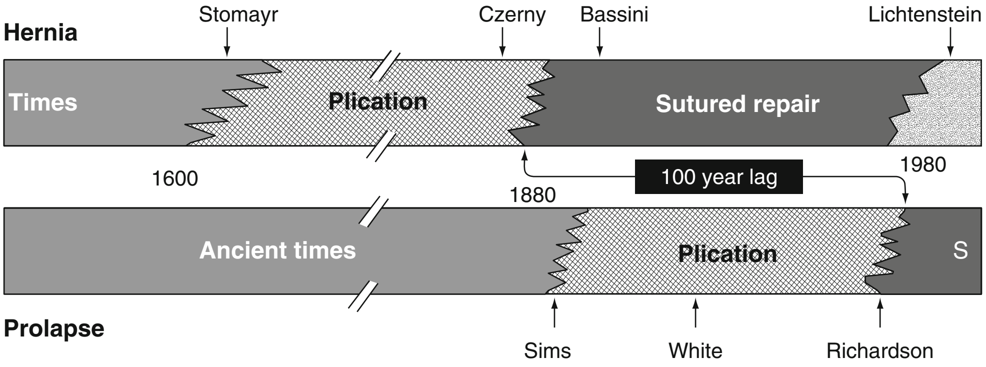

Fig. 3.2 The timelines highlight how gynecologists began empiric plication just as general surgeons abandoned the concept as inherently flawed. Bassini's description of a curative operation ended attempts to control hernia bulges by scarifying or plicating the overlying external oblique aponeurosis. White described an analogous "site-specific" repair for cystocele just 20 years after Bassini, but his concept languished until Richardson's landmark studies half a century later. Gynecologists now lagged herniologists by 100 years

The Era of Anatomic Discovery

The third era of hernia surgery was driven by the anatomic discoveries of the eighteenth and nineteenth centuries.[66] In 1804, Astley Cooper reported that hernia arose secondary to a defect in the transversalis fascia. Cooper further showed that there were two sites of tearing.

- Firstly, there were intrinsic tears within the main body of the transversalis fascia.
- Secondly, the entire fascia transversalis was often avulsed from its normal skeletal attachment to Cooper's ligament and the adjacent suprapubic ramus.

The net effect of these tears was to disrupt the floor of the inguinal canal. In this regard, hernia is obviously analogous to prolapse – which also has tears within the intrinsic fascia and avulsions of the extrinsic fascia from the arcus tendineus on the pelvic sidewall.[46,67]

Following Cooper's discovery that tears in fascia transversalis disrupted the floor of the inguinal canal, *general surgeons* now had a valid understanding of the mechanical factors underlying hernia formation. However, they were unable to exploit this knowledge, because any attempt to enter the inguinal canal was beset with surgical misadventure. *Gynecologists* made no real progress during this era.

The Era of Suture Repair Under Tension

The fourth era of hernia surgery began in 1887, when Geordio Bassini described how "site-specific" tears in fascia transversalis could be identified and repaired. The basic repair was further bolstered by suturing the conjoint tendon and transversalis fascia under tension to the inguinal ligament[23,66] (Fig 3.3a). Modern hernia surgery was born. Looking at the timelines, *hernia surgeons* now understood the mechanical aspects of hernia pathogenesis, and had developed a curative operation (with an operative success rate of about 65%) (Fig 3.2). Hernia repair by suturing native tissues under tension held sway for 100 years, from 1887 to the mid-1980s. During this time, about 70 variations on Bassini's original technique were described, and operative success rates (in specialized units) crept up to ~90%.

By comparison, George White,[68] a surgeon from rural Georgia, was first to conceive of repairing prolapse by "site-specific" fascial repair of the avulsed endopelvic fascia. He became aware of lateral defects while repairing obstetric tears, and published a clear description of how to do a paravaginal repair in 1909. In reality, White's work was before its time. Gynecologists did not really have the skills or the medical support to do retroperitoneal repairs for prolapse in the pre-transfusion and pre-antibiotic era. White's sentinel concept was soon overshadowed by Howard Kelly's[69] more pragmatic advocacy of plication as an approach better suited to stress incontinence and cystocele management in the early 1900s (Fig 3.2). However, anterior and posterior vaginal colporrhaphy began on a large scale in the 1920s, when a host of very experienced military surgeons returned from World War I.

Unfortunately, White's seminal work remained forgotten, long after transfusion and antibiotics had become routine. Whereas *general surgeons* abandoned palliative plication (in favor of a curative fascial repair) some 140 years ago, *gynecologists* have continued with a palliative operation for cystocele and rectocele.

The Era of Tension-Free Repair with Mesh

The era of tension-free synthetic mesh repair began with a report by Lichtenstein and Amid in 1984.[70] Nylon darning

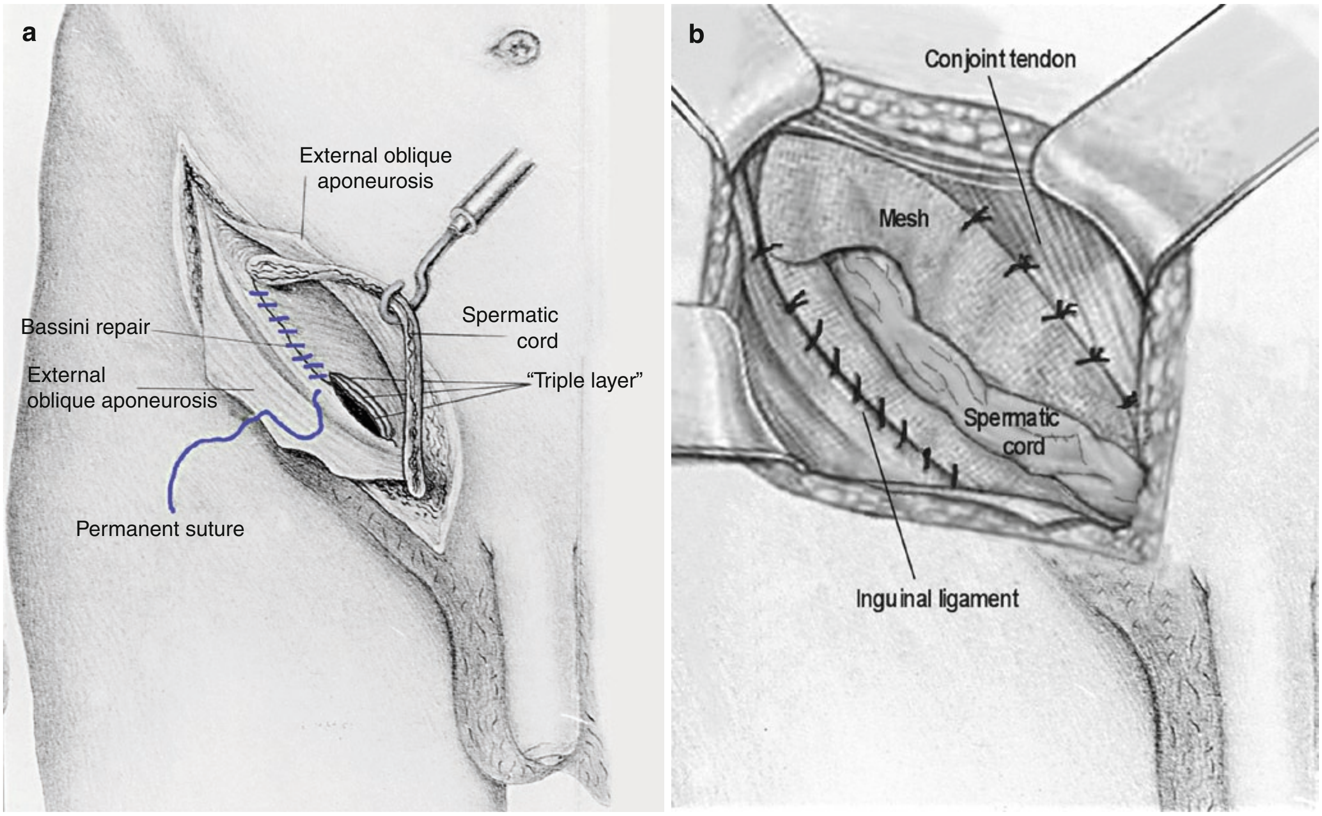

Fig. 3.3 (**a**) The Bassini repair attended to any discernible avulsion in fascia transversalis then bolstered the inguinal canal by sewing a "triple layer" (external oblique aponeurosis, the conjoint tendon, and fascia transversalis) to the inguinal canal, under tension.(**b**) The Lichtenstein tension-free repair is performed by exposing the inguinal canal, mobilizing the spermatic cord and then repairing the damaged fascia transversalis with a mesh onlay

techniques had been used for recurrent hernias since World War II[71-73]; this progressed from darning to the use of a prefabricated nylon weave in the repair of ventral hernia in the 1960s.[74,75] However, the decision to implant synthetic mesh at primary inguinal hernia repair was a serendipitous one. Surgeons at a Los Angeles hernia clinic observed that patients having mesh herniorrhaphy for recurrent hernia had a speedier return to normal activity.[76] They ascribed this reduction in postoperative pain to the avoidance of suture line tension, and therefore elected to repair primary hernias with a simple mesh onlay technique (Fig 3.3b).[70,77,78] This Lichtenstein "tension-free" mesh repair immediately broke through a previous barrier, which had kept recurrence rates for "suture-only" operations above 10%. In hindsight, the reason for these superb results was that mesh prophylactically reinforced any weak adjacent connective tissue. Lichtenstein prosthetic hernioplasty quickly replaced "suture-only" repairs for all but the simplest of hernias.[45,50]

Looking at the timelines, *general surgeons* now had a curative operation that resolved both the mechanical and metabolic components of hernia pathogenesis (Fig 3.4). By comparison, most *gynecologists* in 1984 still believed in Kelly's erroneous fascial attenuation concept, and had not yet begun to question the palliative plication methods described by von Czerny in 1877. The true biomechanics of cystocele and rectocele were not yet understood, and gynecologists remained completely unaware of the secondary

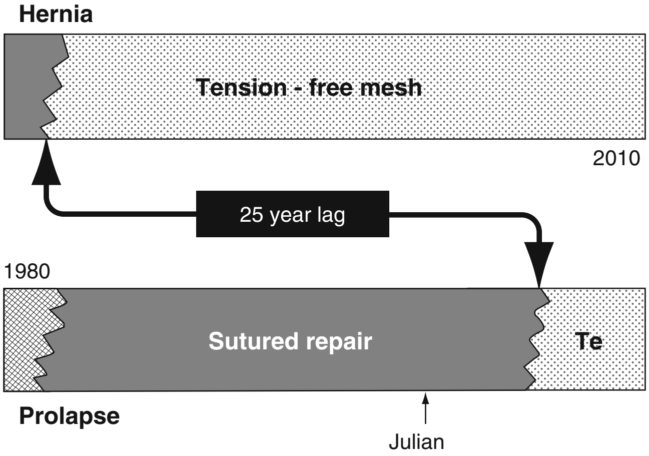

Fig. 3.4 General surgeons progressed from sutured repair under tension to "tension-free" mesh repairs in the mid-1980s. By comparison, most gynecologists were still repairing cystoceles and rectoceles by plication – a technique that herniologists had abandoned a century earlier. Even the elite pelvic reconstructive surgeons who had taken up "site-specific" techniques in the mid-1980s did not move to mesh augmentation until several years after Julian's seminal article of 1996

metabolic factors that fuel so many of the "suture-only" repair failures. In car racing terms, prolapse surgeons were now two laps behind! But change was on the way. Cullen Richardson published his revolutionary concept of site-specific repair in 1976,[79] followed in 1981 by a series of excellent results from abdominal paravaginal repair of cystocele.[80] Even so, Richardson's operation was only the equivalent of Bassini's innovation of 1887. Mesh was introduced for abdominal sacrocolpopexy in the 1980s,[81-83] but only as a way to create a neoligament.

The Era of Laparoscopic Hernia Repair

About a decade after introduction of the Lichtenstein open mesh repair, surgeons began approaching hernias through the laparoscope. The initial method, which was an *intraperitoneal onlay of mesh*, violated the "hernia principles" as they had been discovered to that point, and had a high failure rate. However, this error was soon rectified, and there are now two endoscopic methods which do satisfy the "hernia principles." One is called *transabdominal preperitoneal* (TAPP) and the other is a *totally extraperitoneal* (TEP) repair.[77] Several randomized controlled trials have shown the open and endoscopic procedures to be comparable.[84] Laparoscopic methods have a slightly higher recurrence rate and are much more expensive,[85,86] for the benefit of about 1 day earlier return to full activity.[87] By either technique, *surgeons in special units* have brought failure rates below 2% for primary hernia and perhaps 5% for recurrent hernia.

In prolapse surgery, endoscopy has certainly helped *gynecologists* to visualize the existence and location of the little understood "site-specific" defects on the pelvic sidewall. However, laparoscopic colposacropexy is elitist and expensive, and laparoscopic paravaginal repair perhaps lacks durability in most hands. The transvaginal alternatives of uterosacral/sacrospinous ligament sacropexy and vaginal paravaginal repair seem to offer a more practical solution.[88-90]

The Hernia Principles

For surgery to make an effective transition to the modern era, three major problems had to be solved: bleeding, pain and sepsis.

Prior to the development of techniques for hemostasis and resuscitation, there was an ever present risk of a patient *bleeding to death* on the operating table or at an accident site. In Medieval times, military surgeons controlled amputation bleeding by cauterization, with poor outcomes. The breakthrough was the invention of ligatures by Ambroise Paré in the sixteenth century. However, ligatures remained a mixed blessing until the principles of asepsis were understood. Blood transfusion did not become a realistic option until the 1930s.

The problem of *intraoperative pain* was resolved in the 1840s. Before anesthesia, surgeons had to be as swift as possible, thus largely restricting surgery to amputations and removal of external growths. Anesthesia overcame this dilemma.

- In 1845, Horace Wells, an American dentist, attempted to publicly demonstrate the use of nitrous oxide anesthesia for painless dental extraction. Unfortunately, the gas was incorrectly administered, ruining the effect. Wells was discredited, and died in prison.
- William Morton (another American dentist, and a former partner of Horace Wells) convinced the medical world of the practicality of general anesthesia, by administering ether for removal of a neck tumor at the Massachusetts General Hospital, Boston in 1846.
- In the UK, James Young Simpson began using chloroform in 1847. Anesthesia was given royal sanction when Queen Victoria accepted chloroform for the birth to her eighth child, Prince Leopold, in 1853.

But, despite the rapid spread of anesthesia, surgery was still reserved for emergencies such as amputation, strangulated hernia, compound fracture or obstructed labor – as illustrated by the fact that there were only 333 operations at Massachusetts General Hospital from 1826 to 46.[66]

Major progress against *sepsis* began in the 1867. Historically, wound infection was a major cause of hospital death. Conditions in surgical wards at that time were appalling. Surgeons operated with unwashed hands and dirty instruments, wearing bloodstained operating coats that were seldom washed. Patients then rested in beds with dirty linens that often went unchanged between cases. Many people survived the operation, only to die from gangrene or blood poisoning. Surgical wards were permeated by the smell of putrefaction, giving rise to the belief that infection was caused by "bad air." Joseph Lister, a British surgeon, doubted this explanation. After reading a paper by Louis Pasteur, Lister began sprayng a phenol (carbolic acid) mist during surgery; he also introduced hand washing. Lister's methods quickly reduced infection rates, but Pasteur's "germ theory" was disputed for more than a decade. Nonetheless, by the 1880s, the combination of anesthesia and antisepsis had given birth to the modern era of elective surgery. Hernia was one of the first targets of Victorian surgeons. In contrast, prolapse surgery remained a rarity.

A group of operative rules gradually evolved to deal (initially) with the mechanical elements of failed hernia repair. More recently, these rules have been extended to rationalize the use of tissue augmentation materials. Let us look now at these "hernia principles"– focusing on what they are, how they developed, and what purpose they serve (Table 3.3).

Table 3.3 The "hernia principles"

Traditional principles	
Traditional principles were primarily concerned with dissective technique and gentleness of tissue handling.	
Avoid wound infection	Minimize infection risk through gentle sharp dissection, use of fine suture, no mass pedicle ligation, and strict avoidance of hematoma or seroma. Plication techniques violate these principles
Protect the repair from intra-abdominal pressure	At inguinal hernia surgery, intra-abdominal pressure is contained by ligating the hernial sac at the internal ring and by narrowing the internal/external rings. Analogous strategies at prolapse repair include secure vault re-suspension, high ligation of any enterocoele sac, uterosacral ligament plication with obliteration of a deep cul-de-sac, perineoplasty, and correct alignment of the vaginal axis.
Repair any tears in the investing fascia	Bassini conceived of a genuinely curative hernia operation, by restoring the physiological flap valve mechanism of the normal groin (instead of scarifying the roof of the inguinal canal). The essential dictates were to sew identical tissue within the same layer, using interrupted stitches of permanent suture, without undue suture line tension in any direction. Cystocele and rectocele repair by "site-specific" re-suture of the detached hammocks (instead of scarifying the central fascia) are analogous gynecologic operations. Unfortunately, re-approximation of fatigued native tissues is always likely to create some wound tension, regardless of how well the operation is done.
Re-anchor any torn fascia back onto the skeleton	The fourth traditional principle is to ensure that the investing fascia remains anchored to the axial skeleton. Hernia surgeons solved the problem of frequent inferomedial recurrences by stitching the medial margin of Bassini's repair to Cooper's ligament. Likewise, White and Richardson finally developed a genuinely curative cystocele operation by re-suturing the detached pubocervical septum back onto the white line.
Principles of tension-free mesh repair	
Tension-free hernia repair was first used to reduce suture line tension, but serendipitously delivered the benefit of tissue augmentation.	
Isolate mesh from contact with a hollow viscus	Placing alloplastic mesh in proximity to bowel carries a risk of late entero-cutaneous fistula. Hernia surgeons protect any nearby viscera by using either a composite synthetic mesh (with an adhesive resistant barrier) or a "second-generation" xenograft. The latter strategy has considerable merit in prolapse repair.
Limit bacterial colonization of the mesh	Multifilament polyester mesh forms softer scars, but carries a heightened risk of troublesome infection, if colonized by bacteria. Hence, the use of polyester mesh is undesirable in the vagina. Conversely, polypropylene mesh has partial resistance to bacterial colonization, but forms more erosive scars. Monofilament mesh is reasonably safe in the vagina, but should not be placed into anything other than a clean wound. However, remodeling xenografts are safe in all but the most purulent of wounds.
Minimize the "compliance mismatch" between mesh and native tissue	Mesh weight, stiffness, and construction must suit tissue resilience at the surgical site, and the degree of movement expected at the graft–host interface. In groin hernia, medium weight, macroporous, monofilament polypropylene (Amid type 1) meshes have worked well, but these materials are inherently less suited to the genital tract.
Mesh implant must overlap the defect on all sides	The size and shape mesh must be sufficient to completely cover the hernial defect, and to overlap strong tissue on all sides. As a rule of thumb, hernia surgeons have usually regarded an overlap of 5 cm as sufficient. Attaining the same amount of mesh overlap is not feasible with trocar-driven mesh kits. This limitation may have contributed to the problem of mesh contracture in prolapse repair.
Mesh must be placed in a tension-free manner	Mesh must be shaped to be tension-free when the patient is ambulatory, not just when lying on the operating table. Broadly speaking, this involves keeping the mesh loose (to allow for subsequent contracture), and shaping a slight bowl-like curvature into the center of the implant (to allow for the increase in postural tone when the patient ambulates).

Table 3.3 (continued)

Stabilize against doubling, wrinkling, and undue shrinkage	Interrupted permanent sutures must be placed to prevent subsequent inflammatory reaction from unduly shrinking the mesh or from wrinkling it into a troublesome mass (a "meshoma"). This is not feasible with trocar-driven mesh kits, thus contributing to mesh contracture at prolapse repair.
Choice of mesh must suit surgical objectives	Finally, the exact reason why an implant is being used must be a clearly defined objective. In particular, the surgeon must differentiate between using the mesh as a *neoligament* (in which case, the implant will be subjected to strong static forces) versus using the mesh as an onlay *bolster* or a *bridging graft* (in which case the implant will be subjected to repetitive dynamic forces).

The Traditional Hernia Principles

Avoid Wound Infection

In the pre-Listerian era, hernia surgery had been dogged by sepsis. Even in elective cases, opening the inguinal canal seemed to be a very infection prone, despite the value of carbolic acid spray. Hence, the first of the hernia principles concentrated on minimizing infection risk through optimal tissue handling. Important strategies were: gentle sharp dissection, use of fine suture, no mass pedicle ligation, and the strict avoidance of hematoma or seroma.[66,91-93]

By comparison, many gynecologists doing prolapse repair are still guilty of blunt dissection, rough tissue handling, mass pedicle ligation, often secured with coarse suture and casual hemostasis with undue reliance on packing. All of this favors microbial colonization of the healed wound and a consequent reduction in collagen strength in the final repair.

Protect the Repair from Intra-abdominal Pressure

The second principle, which also evolved during the pre-Listerian era, came from the knowledge that the repaired hernia had to be protected from intra-abdominal forces.[66] In the pre-Victorian era, surgeons attempted to do this by ligating the hernial sac at the external ring, and perhaps sacrificing the testicle. Later, Eduardo Bassini and others evolved a method for high ligation of the sac, together with secure techniques for narrowing the internal and/or external rings.

In prolapse surgery, there are several gynecological equivalents of this second hernia principle:

- The most basic gynecologic equivalent is to prevent postoperative vault prolapse by buttressing apical compartment supports with hysterectomy +/−sacropexy, hysteropexy, or even colpocleisis.
- It is also traditional to stress high ligation of any enterocoele sac (although this maneuver is less important with mesh repairs).
- Any enterocoele repair can be further reenforced by plication of the uterosacral ligament and a Moschcowitz-style obliteration of the cul-de-sac.[94]
- Narrowing a widened urogenital hiatus, to distribute some of the Valsalva forces back onto the pubococcygeus muscles.[95]
- Reestablishing a "hockey stick" vaginal axis, as a means of dissipating any transmitted Valsalva forces against the levator plate.[96]

Repair Tears in the Investing Fascia

The third principle derived from Bassini's recognition that inguinal hernia could be cured by repairing torn transversalis fascia in the floor of the inguinal canal. Dictates were that the surgeon should sew identical tissue within the same layer,[97] using interrupted stitches of permanent suture,[98,99] without undue suture line tension in any direction.[77] Suture line tension compromises blood supply, thus creating substantial postoperative pain and a risk of the approximated structures pulling apart before healing is complete. This fascial repair was then buttressed by sewing a "triple layer" (external oblique aponeurosis, the conjoint tendon, and fascia transversalis) onto the inguinal ligament (Fig 3.3a). Unfortunately, in sewing together structures that do not normally approximate, Bassini's operation invariably led to the suture line tension he sought to avoid – regardless of the technical skill with which the fascial repair had been done.

A gynecologic equivalent of the third principle is reattaching the pubocervical or rectovaginal septae back onto the pericervical ring at "site-specific" cystocele or rectocele repair. Reefing together ill-defined "white stuff" under tension at anterior colporrhaphy or grossly constricting the vaginal canal to contain a rectocele violates the third hernia principle.

Re-anchor the Fascial Hammock Back onto Skeleton

The fourth principle is another legacy of the Bassini's landmark advances. Stabilizing the canal roof by stitching the conjoint tendon to the inguinal ligament (and hence the pelvic girdle) serendipitously prevented lateral hernia recurrence. However, inferomedial recurrences remained a problem. This technical inadequacy was circumvented by re-anchoring the medial margin of Bassini's repair to the superior pubic ramus (usually via Cooper's ligament).

Gynecologic equivalents of the fourth principle are:

- Any some form of colpopexy that re-anchors the vaginal vault back onto the uterosacral ligaments, the sacrospinous ligaments, or the sacral promontory (see Section "Postero-Apical Compartment").
- Sewing an avulsed lateral margin of pubocervical or rectovaginal fascia back onto the parietal fascia of obturator internus or levator ani muscle (see Section "Anterior Compartment").

Note that repair of a paravaginal defect is really an adherence to the fourth principle, and repair of a superior defect is really an adherence to the third principle.

Principles for Synthetic Mesh Hernia Repair

By the middle of the twentieth century, the concepts of accurately repairing all "site-specific" fascial defects by gentle technique were "set in stone." The need to ensure that the abdominal wall connective tissues remained anchored to the axial skeleton was also well appreciated. These traditional hernia principles have long formed the background of surgical training, providing an arena in which junior surgeons learn fine dissective skills.[91]

Although these maneuvers were broadly successful, excessive wound tension sometimes impeded healing, thus creating a "glass ceiling" for surgical success rates. Relaxing incisions were introduced in 1892, but could only reduce (rather than eliminate) wound tension at sutured herniorrhaphy.[100] Some 25 years ago, surgeons discovered that the best way to resolve the problem of wound tension was through the use of a mesh implant. This strategy automatically reinforced any weakness in the adjacent connective tissues. Mesh implants also made repair of the mechanical defect quicker, easier [70,101] and more cost effective.[85-87,102]

Isolate Mesh from Contact with a Hollow Viscus

One of the first lessons learned in the use of synthetic mesh was that placing alloplastic mesh too close to a hollow viscus risked late entero-cutaneous fistula.[103-106] Hernia surgeons now circumvent this obstacle with either a composite synthetic mesh (incorporating a nonadhesive barrier) or a "second-generation" biological implant[107-109] (see Chap. 10, Sects. 1.1 and 1.2). The use of collagen coating of polypropylene mesh at prolapse repair does not provide secure protection against bladder or bowel erosion.

Limit Bacterial Colonization of the Mesh

By forming a slime layer, bacteria can adhere to any type of alloplastic material.[110,111] Dormant organisms can subsequently reactivate, producing a mesh-related sepsis months or even years after implantation.[30,112] While all synthetic implants are susceptible, infection rates and severity are greatest with Amid classes II and III meshes. In an audit of the four hernia materials used at Tufts University School of Medicine from 1985 to 1994, Mersilene® (an uncoated multifilament polyester mesh) had the most complications per patient (4.7 vs 1.4–2.3; $p < .002$), the highest incidence of enterocutaneous fistula (16% vs 0%–2%; $p < .001$), more frequent surgical site infections (16% vs 0–6%; $p < .05$), and the highest hernia recurrence rate (34% vs 10–14%; $p < .05$).[103] Subsequent surgeons who did not heed Leber's warning have also reported enterocutaneous fistula and chronic sinus formation with Mersilene® mesh.[105,106] This differential arises because macrophages and natural killer cells (9–20 μm) are too large to penetrate the microporous gaps of a class II mesh or to infiltrate the spaces between multifilamentous fibers of a type III mesh. Thus, any bacteria (<1 μm) that disperse within the small interstices between fibers escape phagocytosis.[11]

The potential for mesh infection influences what type of implant can be safely used in prolapse and incontinence surgery:

- Given the troublesome septic sequelae attending the use of multifilamentous mesh, even in relatively sterile hernia incisions, placing polyester mesh into a potentially contaminated vaginal repair would seem unwise. Any infection is likely to progress to a severe granulomatous reaction, thus necessitating removal of the entire implant.[113-115]
- Infection of a polypropylene mesh will usually settle on antibiotics, without the need for mesh removal.[116] Even so, the trocar-guided prolapse repair kits are still troubled by substantial mesh morbidity rates.[117] If an intestinal cavity has been entered during attempted rectocele repair, polypropylene mesh should definitely not be placed.
- "Second-generation" biomesh has been used successfully in overtly infected abdominal wall wounds (e.g., to close large myo-aponeurotic defects complicating fecal peritonitis).[118-120] As such, it is permissible to complete a postero-apical compartment reconstruction with a tissue inductive biomesh, even if fecal contamination has occurred. In fact, many surgeons now routinely employ porcine small intestinal submucosa (Surgisis®, Cook Surgical, Bloomfield, IN) as an interposition graft in rectovaginal fistula repair. Obviously, the wound should be vigorously irrigated with normal saline, before closing.

Minimize the "Compliance Mismatch" Between Mesh and Native Tissue

Mesh weight, stiffness, and construction must suit tissue resilience at the surgical site, and the degree of movement expected at the graft–host interface. Multifilament polyester meshes are "wettable" – leading to softer scar reactions; however, polyester mesh has fallen out of favor because of a heightened risk of granulomatous infection, if colonized by bacteria. In contrast, monofilament polypropylene mesh is "non-wettable" – leading to harder scar formation, but a reduced susceptibility to granuloma or chronic wound sinus formation.[103,112] Medium weight macroporous monofilament polypropylene meshes have worked well in groin hernia, but their torsional rigidity often causes undue abdominal wall stiffness in ventral hernia.[121] For prolapse surgery, mesh weight has been reduced from ~150 g/m^2 for a traditional heavy weight hernia mesh to ~50 g/m^2 for Gynemesh® (Ethicon, Somerville, NJ). However, studies to date have not found lower morbidity with further reductions in mesh weight (to ~30 g/m^2).[122-124] Failure rate may also be higher.[125,126]

Mesh Implant Must Overlap the Defect on All Sides

Mesh size and shape must completely cover the hernial defect and overlap strong tissue on all sides. As a rule of thumb, hernia surgeons have usually regarded an overlap of 5 cm as sufficient.[127-131] Attaining an equivalent overlap of synthetic mesh with the trocar-driven prolapse repair kits is not possible. This technical limitation has contributed to the problem of mesh contracture. When using a tissue inductive biomesh, an appropriate overlap of the donor tissue is crucial (see Chap. 10).

Mesh Must Be Placed in a "Tension-Free" Manner

A key safety factor is that any mesh must be shaped to be "tension-free" when the patient is ambulatory, not just when lying on the operating table.[129-131] Broadly speaking, this involves keeping the mesh loose (to allow for ~30% subsequent contracture[127]), and shaping a slight bowl-like curvature into the mesh (to allow for increased postural tone when the patient is ambulatory). In prolapse repair, it is just as important to place any synthetic or biological implant loosely enough to allow for the extra hammock tension created by standing erect.[132]

Stabilize Against Doubling, Wrinkling, and Undue Shrinkage

All synthetic mesh implants evoke a strong foreign body reaction that continues for many years.[133,134] General surgeons learned through bitter experience to anchor mesh with interrupted *permanent* sutures, to prevent subsequent inflammatory reaction from contracting the mesh into a troublesome mass (a "meshoma").[110] Absorbable and delayed absorbable sutures are not adequate for this task.

Gynecologists have been slow to grasp the concept that mesh must be permanently secured against migration in any direction.[135] The one notable exception to this rule is placement of long, narrow mid-urethral tapes by closed technique. Unfortunately, many gynecologists have confused the exception with the rule, and have misinterpreted the term "tension-free" to mean "not suturing mesh in place." This is a serious error, which will create needless complications for those who place unsecured mesh sheets at open vaginal surgery. Even with trocar-guided prolapse repair, postoperative mesh shrinkage remains a real problem. This arises because the transobturator arms resist contraction in a mediolateral, but not an anteroposterior direction. For example, sonographic measurements of mesh shrinkage in the first 6 weeks after unsecured vaginal polypropylene mesh repair showed an average anteroposterior shrinkage of 57% for cystocele and 46% for rectocele prostheses.[136] Placing synthetic mesh without secure, lasting anchorage breaks one of the very basic hernia principles.

Principles for Biological Mesh Hernia Repair

Alloplastic suture materials were developed in the 1940s, but their use as reinforcing prosthetics was initially shunned by hernia surgeons. Attitudes changed in 1958, when Usher[137] cured large ventral hernias by tensionless preperitoneal placement of Marlex® mesh (a medium weight macroporous polypropylene made by CR Bard Inc, Murray Hill, NJ). But surgeons of the 1970s initially preferred uncoated polyester implants (Mersilene®, Ethicon, Somerville, NJ; Dacron®, DuPont, Kinston, NC), because of their superior handling properties and softer scar formation.[74,75] Unfortunately, fibroblast and vascular ingrowth are restricted by their microporous and/or multifilamentous construction; hence, Amid classes II (microporous) and III (multifilament) meshes tend to encapsulate within a mini-bursa, creating a potentially weak anchorage site. Their heightened susceptibility to chronic sepsis was a second problem (see Chap. 10).

Herniologists soon switched to Amid class I (macroporous monofilament) implants because of their infection resistance and more robust healing.[138] Macroporous monofilament mesh is more readily penetrated by vascular and fibroblast ingrowth; scar maturation later strangles the areas of neovascularization. Provided there is no undue graft-tissue motion,[139] polypropylene mesh is generally incorporated into a felt-like collagenous band that is strongly attached to adjacent host tissues.[11,140,141] However, there is a downside. Amid class I meshes are torsionally rigid and form more abrasive scars.[121]

Compliance mismatch is well tolerated by the relatively static tissues of the groin. But in the more mobile tissues of the anterior wall, constant shearing of tissue across an abrasive mesh sets up a "cheese grater" effect – creating severe cicatrization, mesh exposure, and a risk of fistula formation.

Searching for a less cicatrizing material, manufacturers in the early 1990s deliberately "leatherized" various cadaveric and animal grafts, in the hope of producing a permanent but "more natural" implant. Outcome proved to be disappointing, with wound problems and poor cure rates. With the wisdom of hindsight, the reason for these seemingly paradoxical results is obvious. In vivo, any denatured collagen – whether of endogenous or exogenous origin – is seen by the host immune system as "dead tissue," and thus subjected to an intense biodegradation reaction (i.e., encapsulation and enzymatic autolysis) (see Chap. 10).

Much of the adverse healing pattern seen with solid sheets of "first-generation" biomesh occurs because the host immune response cannot penetrate these dense, collapsed collagen matrices. The tendency to seroma formation was later reduced by fenestrating the original product. While durability of Pelvicol Soft® (CR Bard Inc, Murray Hill, NJ) as a stand-alone implant remains suspect, this long-lasting biomaterial has been combined (somewhat unsuccessfully) with polypropylene to reduce host inflammatory response (Avaulta®).[142]

Surgical implants are designed to re-attach an area of avulsed connective tissue back onto the body wall by soft tissue ingrowth. When considering tissue augmentation, it is intuitive to select an inert permanent material. However, all synthetic meshes and crosslinked biologicals evoke a foreign body inflammatory reaction, meaning that there is always a fine line between benefit and morbidity.[140] Scientists later recognized the potential for a bioabsorbable prosthesis to deliver a permanent repair, through a tissue engineering process known as constructive remodeling[107-109,143-145] (see Chap. 10, Sect. 3.3). "Second-generation" bioabsorbable scaffolds are noninflammatory, infection resistant[146,147] and specifically designed to disappear from the wound once healing is complete.[148] Hence, there is no potential for cicatrization or graft erosion,[62] and wound pain is significantly reduced.[109] Key points in the tissue inductive process are ensuring preservation of collagen structure and matrix molecules during manufacture[149,150]; biodesign of a scaffold that will hold the wound in apposition long enough for constructive remodeling to lay down mature collagen (typically, about 3–5 months)[145,151]; overlapping the implant across the layer from which host cell repopulation is sought[12,132]; and exposing the graft to suitable mechanical stresses during wound healing.[64,65,152,153] The operation of these tissue engineering variables is further modified by host metabolic status – as reflected by age, nutrition, androgen status, and the presence of any dysregulatory factors (e.g., diabetes, autoimmune connective tissue disease)[62,144]

Should Gynecologists Adopt These Hernia Principles?

These "hernia principles" appear relevant to pelvic reconstructive surgery, at least at a conceptual level. But we cannot directly extrapolate the choice of materials, from hernia to prolapse.[88,154-160] The vagina is not the abdomen:

- *In the groin*, mesh is implanted through a sterile environment, between two tough and highly collagenized aponeurotic layers, where it lies 5–10 cm deep to body surface. There is minimal tissue-on-tissue movement, and the mesh is well separated from intra-abdominal hollow viscera.
- *In the vagina*, mesh is implanted through a contaminated environment, between a basement membrane and a fragile layer of smooth muscle, just ½ cm deep to vaginal mucosa. This is an area of maximal tissue-on-tissue movement. Finally, the implantation site is immediately adjacent to the bladder, ileum, and rectum.

Recognizing the "compliance mismatch" differences between the groin and vagina is especially important. To this end, the precise objective for placing the implant must be clearly defined. The pelvic reconstructive surgeon must differentiate between using the mesh as a *neoligament* (in which case the implant will be subjected to strong static forces) versus using the mesh as an *onlay bolster* or a *bridging graft* (in which case the implant will be subjected to repetitive dynamic forces)[90]

Gynecologists have traditionally regarded cystocele, rectocele, enterocoele, and vault inversion as four discrete entities. However, this view is dated.

- From a surgical anatomy perspective, pelvic connective tissues are organized into two semi-independent systems – the anterior (bladder) and postero-apical (rectal and uterine) compartments. These two compartments intersect like a flag and flagpole (Fig 3.5). The *anterior hammock* is vital to urinary continence, but has no major supportive role for the vagina as a whole.[161] Conversely, the *postero-apical* connective tissue both suspends the pelvic organs and partitions the vagina from the rectum.[89]
- From an engineering perspective, the pelvic connective tissues seem to constitute an "integrated structure," meaning that the integrity of one compartment depends on the other parts of the system being intact.[162] Thus, support failure within the anterior and postero-apical compartments is highly correlated.[36,89,163,164]

Patients usually present with overt support failure in one segment and incipient weakness in adjacent sites. Paradoxically, despite marked differences in their clinical prominence, both

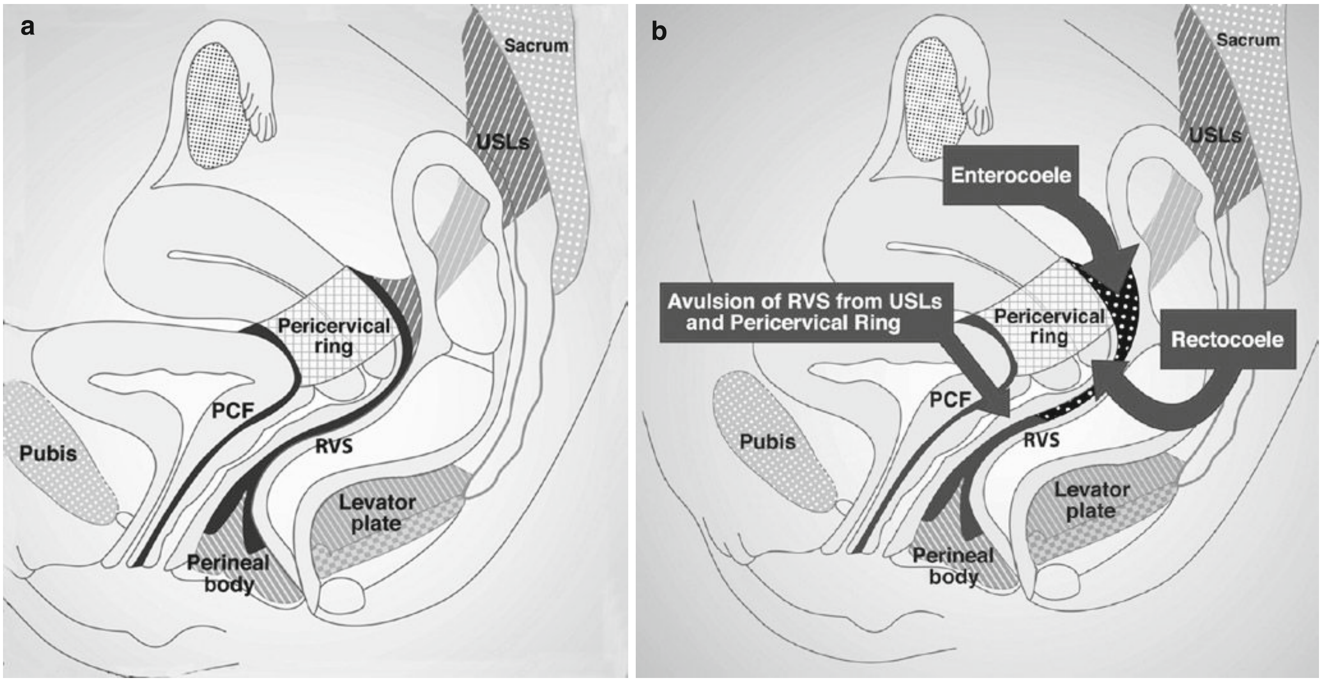

Fig. 3.5 (**a**) A sagittal section of female pelvis, showing the vaginal suspensory axis and anterior vaginal hammock The *postero-superior vaginal suspensory axis* is a continuous sheet of strong connective tissue, running from the sacral periosteum, through the uterosacral ligaments (USLs), onto the pericervical ring, and down through the rectovaginal septum (RVS), to insert into the apex of perineal body. When this is intact, bowel motions are guided smoothly through the pelvis and easily out the anus. When torn, pelvic dragging discomfort and obstructive defecation become a problem. The *anterior suspensory hammock* is formed by the pubocervical fascia (PCF), as it runs caudad to insert into the perineal membrane (urogenital diaphragm). Obstetric forces typically tear the fascia in the mid-pelvis. Fracture above or below the pericervical ring has differing clinical consequences. (**b**) A diagram showing how laceration of the uterosacral ligaments *above* the pericervical ring leads to uterine descensus, while avulsion of the rectovaginal septum *below* the pericervical ring permits herniation of ileum, sigmoid or rectum into the vaginal lumen

dominant and incipient support defects are of almost equal importance to the reconstructive gynecologist. The fascial supports at the secondary sites may well be strong enough to maintain the *status quo*, but may be too damaged to resist the new force vectors created when an adjacent vaginal segment is re-suspended. Not repairing an area of incipient weakness in such circumstances sews the seeds of early failure – often within months. In the words of Wayne Baden,[165] the prudent surgeon will always "leave the entire tract intact," or face an unacceptable risk of early postoperative bladder, vault, or rectal prolapse.

Postero-Apical Compartment

As stated, the *vaginal suspensory axis* suspends the vaginal apex and partitions the vagina from the cul-de-sac and rectum. When intact, this vaginal suspensory axis forms a membrane that guides feces efficiently through the pelvis and out the anus. The proximate cause of recto-enterocoele is a "site-specific tear" in the vaginal suspensory axis – creating *suspensory failure* if the injury occurs above the pericervical ring and *partition failure* if damage occurs more distally[166] (Fig 3.5b).

An adequate recto-enterocoele repair can be done by mobilizing the distally displaced rectovaginal septum and re-suturing it to the pericervical ring.[89] However, given that torn endopelvic connective tissues undergo a slow but relentless deterioration in collagen quality, use of an appropriate tissue augmentation material is more in accordance with modern hernia principles. If mesh is to be used, the surgeon must satisfy two different goals:

- *Re-attachment of the vaginal fascia onto the axial skeleton (via the uterosacral ligament insertion into the sacral hollow):* Mesh used for this task must act as a "neoligament," for which tensile strength is the dominant consideration. Polypropylene is the strongest available material, but morbidity potential must be balanced against the extra tensile strength gained. As can be deduced from the hernia principles, using synthetic mesh as a suspensory strut at static sites (e.g., spanning the mid-pelvis or traversing the pararectal space) is unlikely to cause compliance mismatch. Conversely, filling the rectovaginal space (an area of high tissue-on-tissue mobility) with polypropylene risks erosion or dyspareunia (see Chap. 10, Sects. 3.3 and 3.4). My philosophy is to rely on a "second-generation" remodeling biomesh, except in the presence of extreme failure hazard.

- *Closure of any low-pressure zone within the postero-apical compartment:* This needs a bridging graft, not a strut. The graft material must be strong, but not excessively so. The prime considerations are preservation of tissue flexibility and a low erosion or pain risk. A "second-generation" remodeling biomesh will almost always be strong enough for this role[62,140] (see Chap. 10, Sect. 4.4).

Effective repair of postero-apical compartment prolapse requires that fascial integrity be reestablished in two different planes.

- In the *sagittal plane*, fascial continuity must be restored from the sacral periosteum, through the uterosacral ligaments, onto the pericervical ring, down the rectovaginal septum, and into the perineal body (Fig 3.5). Historically, this has been most effectively done by threading a narrow ribbon of polypropylene through the rectovaginal space, from sacral promontory to perineal body (abdominal sacrocolpopexy).[81,167] However, transvaginal placement of a remodeling biomesh has the potential to deliver even better performance than abdominal sacrocolpopexy, by a cheaper and less invasive technique.[88,90]
- In the *coronal plane*, transverse avulsion of posterior compartment fascia usually extends from sidewall to sidewall. Restoration of normal anatomy requires that fascial continuity be established from the ischial spines and lower margin of sacrospinous ligament, down the white lines,[168] to the distally retracted edge of the rectovaginal septum (Fig 3.6). This is difficult to do from an abdominal approach, because it is near impossible to synchronously open the rectovaginal and both pararectal spaces from above. Conversely, the vaginal surgeon can readily expose all three spaces in the coronal plane. This provides superb access for placing two pairs of stay points (sacrospinous ligaments laterally and *extraperitoneal* margin of uterosacral ligaments at the top of the rectovaginal space).[88-90] These stay sutures then secure a pre-cut bridging graft of porcine small intestinal submucosa (Posterior Pelvic Floor Graft®, Cook Medical Incorporated, Bloomington, IN) to the sacral hollow at about S_3 level[88]

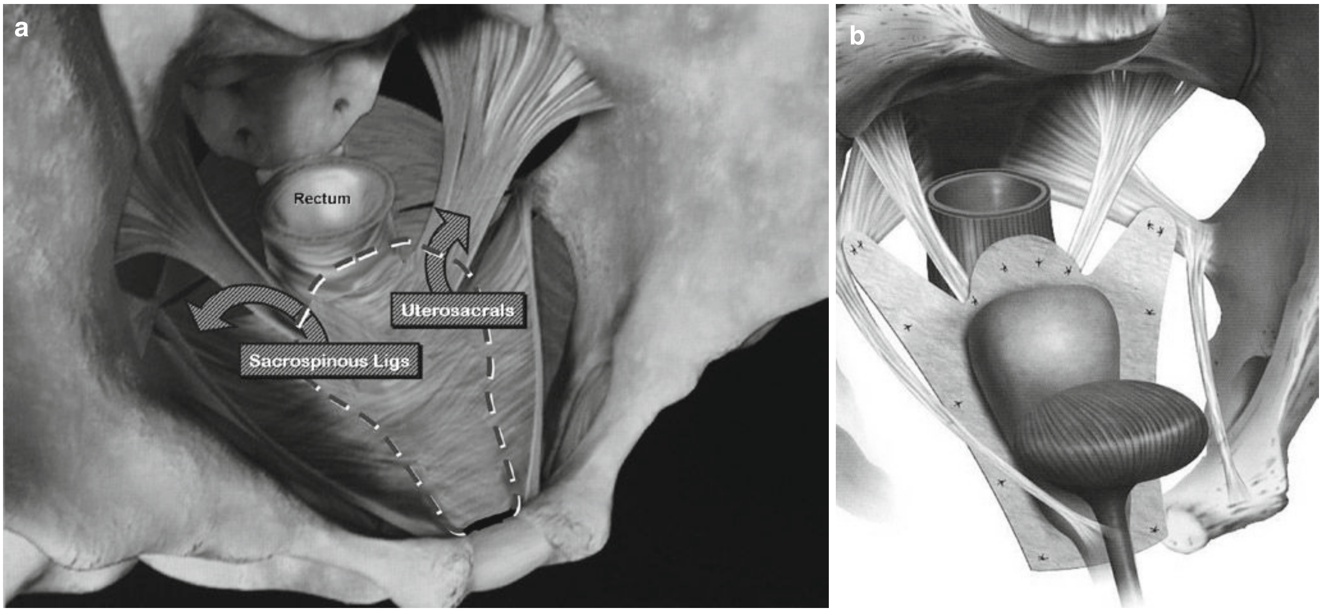

Fig. 3.6 **(a)** The postero-apical compartment fascia in coronal section showing how the uterosacral ligaments extend caudally as the lateral vaginal septae. These septae subdivide the posterior compartment fascia into the rectovaginal septum (centrally) and the pararectal spaces (laterally). The position occupied by the vagina (i.e., the rectovaginal septum) is indicated by the dashed line. When intact, this posterior compartment fascia partitions the rectum from the genitourinary system, and guides the stool through the pelvis. Obstetric trauma usually lacerates this partition from sidewall (ATFP) to sidewall, creating a defect that extends across both rectovaginal and pararectal spaces. Such trauma disrupts both local anatomic and the mechanics of defecation. In repairing a recto-enterocoele, resolving the obstructed defecation is just as important as controlling the prolapse bulge. **(b)** A pre-shaped posterior compartment porcine submucosal graft (Surgisis® Biodesign™ Posterior Pelvic Floor Graft, Cook Medical, Bloomington, IN), which allows the surgeon to perform a sacrocolpopexy from below. Two pairs of stay sutures secure this repair device to the sacral hollow (via the extraperitoneal margin of uterosacral ligament insertions) and to the sacrospinous ligaments (in the pararectal spaces). The graft is then tensioned in all directions by tacking it to levator fascia (laterally) and the apex of perineal body (distally)

Anterior Compartment

Despite cystocoele repair being among the commonest operations in gynecology,[169] success rates and long-term repair durability are poorly described.[170] Case series on anterior colporrhaphy generally reported recurrence rates in the 0–30% range; however, subsequent randomized control trials show anatomic failure rates to be much higher than previously believed. Sand[171] had a 43% recurrence at 12 months, and Weber[172] had a 61% objective failure rate at 2 years. Moreover, the tails of the Kaplan-Meier curves were still falling at study conclusion (27 months). That is not to say that every single anterior repair is unhelpful. Colporrhaphy is a simple and reasonably effective strategy for short-term relief of bulge discomfort, and a proportion of plication cystocoele repairs do prove durable through the formation of a nonspecific scar plate beneath the vesical neck and bladder base.[12,173,174] Nonetheless, anterior colporrhaphy is clearly not reliable enough to be the generic standard for cystocele repair.

This unsatisfactory state of affairs is predicted by the hernia principles. The urethra and bladder are suspended by a trapezoid-shaped sheet of endopelvic fascia that is tightly strung to the cervix (above), the pelvic sidewalls (laterally), and the pubic bones (below). As such, the anterior hammock functions like a trampoline, providing all direction support to the urethra and bladder (Fig 3.7a). Gynecologists have traditionally believed that the central fascia of this suspensory hammock stretches after childbirth, thus forming the bulge of a cystocele. In reality, pelvic fascia is like canvas – it does not stretch, but it will tear at pre-determined weak points. As a matter of engineering principle, these weak points always lie at top and side, not centrally. Fascial tearing along the peripheral margins turns the trampoline to a trapdoor, creating a central bulge (Fig 3.7b). However, attempts to control the bulge by a plicative thickening of the sagging (but intact) central fascia do not meet the dictates of the hernia principles. Formation of a rotatory cystocele has three elements: an apical defect, a lateral defect on at least one side, and a fulcrum about which rotation can occur. This fulcrum can be located at either the urogenital diaphragm (creating diffuse descent of the entire anterior vaginal wall and a tendency to stress urinary incontinence), or the vesical neck (creating a high cystocele and a tendency to voiding dysfunction). Correcting a cystocele in accordance with these biomechanical principles mandates "site-specific" repair of the causative fascial avulsions, either with permanent suture or by placement of a mesh bolster.

It is self-evident that an operative strategy which ignores the primary mechanical events causing the prolapse must inevitably lack long-term reliability. Reconstructive surgeons are now turning away from traditional anterior and posterior colporrhaphy. Unfortunately, the pelvic sidewall is a surgically

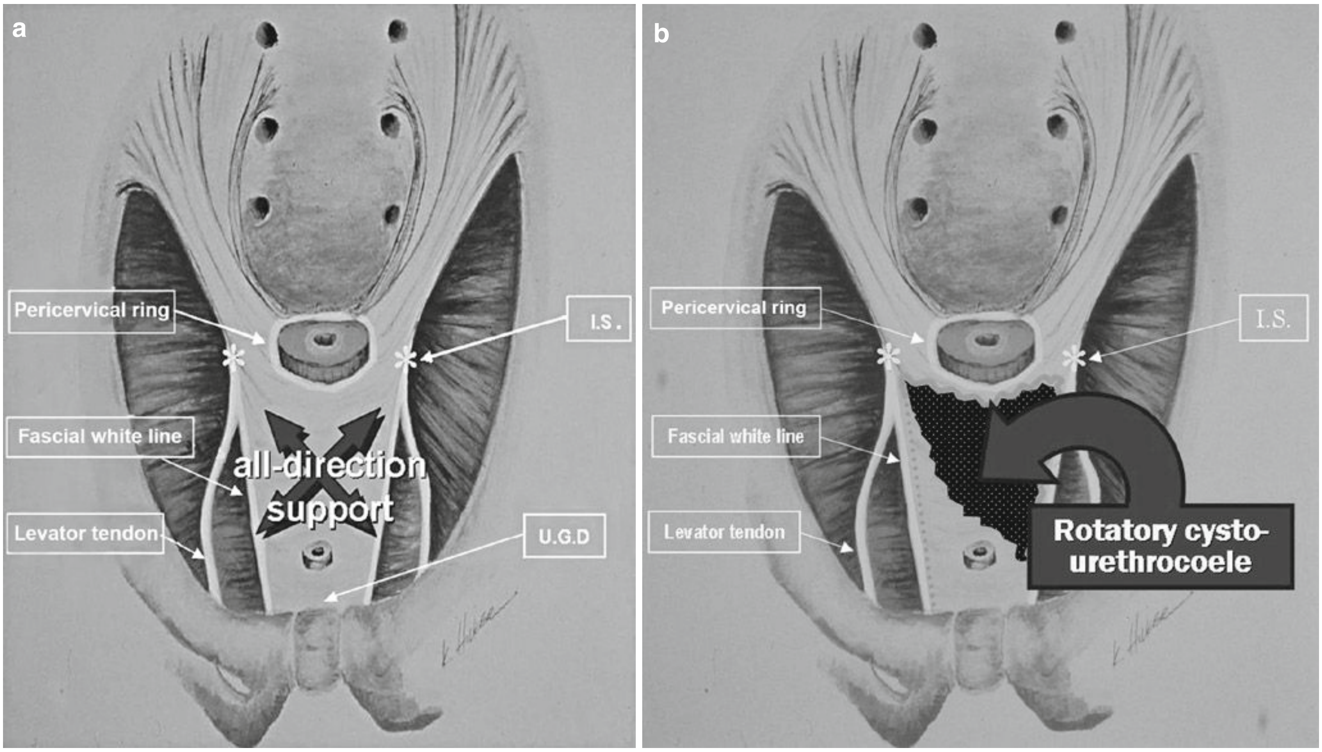

Fig. 3.7 **(a)** The "flag" is a highly specialized fascial diaphragm which gives "all direction" support, like a trampoline. However, there are *lines* of weakness along the *top* and *lateral* margins. (**b**) If torn, a large defect develops. Net effect is that the "trampoline" is turned into a "trapdoor"

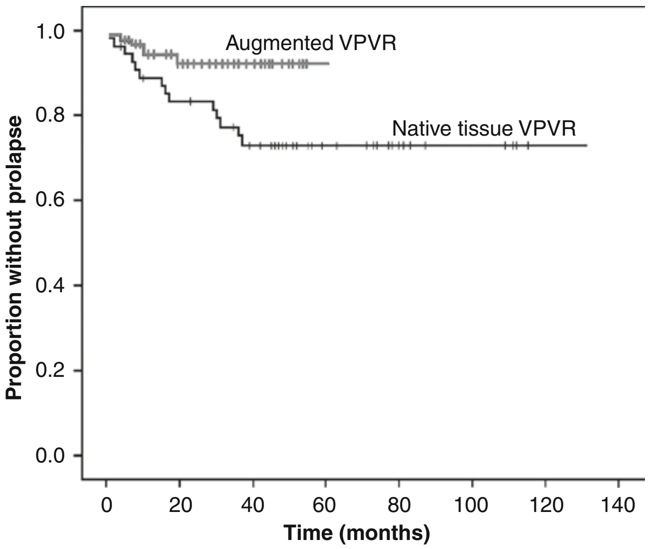

Fig. 3.8 Ten-year Kaplan-Meier survival analysis data comparing augmented versus native tissue VPVR. The use of any form of augmentation was significantly better than suture-only repair (logrank $\chi^2 = 4.48$, *p*-value $= 0.0343 < 0.05$). Late failures continued for longer in the native tissue group, suggesting a greater impact of either suture line tension or connective tissue weakness when a biomaterial was not used. Nonetheless, both curves eventually flattened – augmented repair at about 19 months and sutured VPVR at about 38 months. These results suggest that the remaining women had obtained a durable cystocoele

hazardous area, unfamiliar to many generalists. To circumvent this difficulty, several Medical Device companies have marketed surgical kits that allow surgeons to more easily place plastic mesh implants into the sagging vaginal walls, using long curved trocars. These devices certainly repair the prolapse, but their popularity has been market (not evidence) driven. Advocacy for these methods was based mainly on the successful use of polypropylene slings at relatively static genital sites, and the proven superiority of prosthetic hernioplasty over non-augmented suture repair. Unfortunately, there is still a paucity of reliable safety and efficacy data. Reported morbidity rates are now creeping towards ~20%. Cautionary articles have been issued by a virtual "Who's Who" of urogynecology – from UCLA, University of Michigan, Baylor College of Medicine, McMaster University, University of Milan, Karolinska Institute, Cleveland Clinic, Mayo Clinic, Long Beach Memorial Hospital, West of Scotland Study Group and two IUGA Past Presidents.[113,117,135,154-160,175-177] There has also been a recent alert from the American Food and Drug Administration (http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm142636.htm) warning that, over the past 3 years, FDA has received >1,000 mesh manufacturer reports of complications associated with these minimally invasive – but not necessarily minimally harmful – devices.

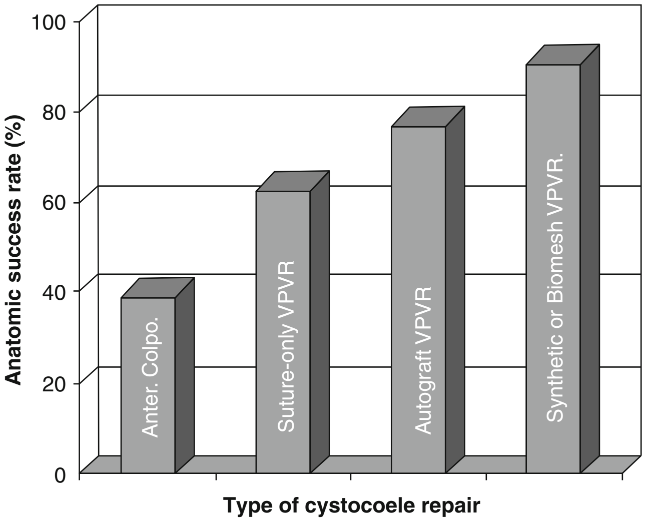

Fig. 3.9 Weber's[109] results for anterior repair are compared in a bar graph with the various techniques for VPVR,[6] ranked in approximate accordance with their conformity to the "hernia principles." Success rates for cystocoele repair showed stepwise improvement from *left* to *right*

My preference in the anterior compartment has been for the use of "second-generation" biomesh. In a database of 219 cystocele repairs over an 11-year period,[12,46] augmented vaginal paravaginal repair outperformed native VPVR by a margin of 28.6% (91.2% versus 62.6%; logrank $\chi^2 = 8.9$, *p*-value $= 0.0028 < 0.05$). Both techniques were genuinely curative of cystocele, as evidenced by an absolute flattening of the Kaplan-Meier curves at 40 months (Fig 3.8). However, Cox proportional hazards modeling showed that use of a tissue inductive xenograft reduced the risk of repair failure by a 69.4% (CI = 26.9–86.9%). Functional outcomes in both groups were also excellent. Perioperative complication rate was 4.7%, with no mesh-related morbidity. On subgroup analysis, VPVR with bridging graft of Surgisis® outperformed the "suture-only" and vaginal autograft techniques (98% vs 84% vs 65%). On subgroup analysis, success rates improved incrementally with increasing adherence to the "hernia principles" (Fig 3.9).

Conclusion

Pelvic floor disorders affect about half of the female population, and represent one of the major problems of later life. Twenty percent of elective gynecological surgery is done for prolapse,[169] and this figure will increase as the population ages. Worldwide, prolapse and incontinence cost society about US$100 billion per year[178-180]; this compares to what is spent on gynecological cancer. Traditional colporrhaphy is based upon flawed concepts from the 1920s. Plication repair does not address the true sites of fascial damage, and therefore has an unacceptable failure rate – irrespective of surgical skill or operative technique. Given the astounding prevalence and high cost burden of pelvic organ prolapse, society can no longer afford to persist with such suboptimal therapies.

Even "site-specific" prolapse repairs with permanent suture are not truly reliable. Although paravaginal repair of cystocele satisfies modern biomechanical principles, any form of native tissue re-suture still has ~30% failure rate.[46,181] Gynecologists must acknowledge that symptomatic prolapse reflects a combination of primary fascial tearing and secondary collagen weakness. As such, the lessons from herniology are very relevant. The "hernia principles" suggest that an optimal prolapse surgery should combine "site-specific" fascial repair with a suitable implant to bolster weakened regional connective tissue. Trocar-driven mesh kits make this task technically easier for the surgeon, but carry significant risk of mesh morbidity. Moreover, these mesh kits are very expensive. An alternative solution lies with placing bioabsorbable xenografts at open transvaginal surgery. This approach satisfies all of the modern hernia principles, and delivers "gold standard" cure rates without mesh morbidity.

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4

Diagnosis of Uterovaginal Support

S. Robert Kovac

Vaginal prolapse has been a common malady for over 2,000 years usually treated by a variety of pessaries and chemical concoctions. Surgical attempts to cure this condition started in the late 1800s and early 1900s. For the past 130 years, gynecologists have accepted the belief that vaginal defects causing anterior and posterior vaginal prolapse were either from midline stretching of the pubocervical fascia as assumed by Kelly[1] in 1913 or lateral or paravaginal injuries as suggested by White[2] in 1909 and Richardson et al.[3] in 1976. However, these theories seem to conflict with each other. Kelly's view was based on the central stretching of supportive tissues, more commonly view as a central or midline defect. Kelly corrected this bulge of the bladder by folding the stretched tissue on itself. White considered the cause of a cystocele to be a mechanical tear of the supportive tissues of the bladder from the sidewall of the vagina. Kelly's operation was based on "getting rid" of the bulge, while White wished to support the anterior vaginal wall.

It was believed that defects of the supportive structures of the bladder and rectum causing vaginal prolapse were associated with vaginal birth. A major shortcoming of the profession was the effect of labor and delivery on the female pelvis and vaginal prolapse had not been fully understood. With clinical observation, there is little doubt that childbirth contributes to the likelihood that clinically symptomatic prolapse will occur. Unfortunately, there was little thought as to how and when in the course of labor the effects of childbirth caused fascial tears, or if the tears occur in a vertical (midline) or lateral (paravaginal) direction. Before the surgeon can decide on what type of repair to perform, it is necessary to understand how cystoceles and rectoceles occur following vaginal birth. Therefore, in order to correct the anatomic cause of vaginal prolapse, surgeons need to understand both the anatomy and how it becomes defective before instituting surgical repairs.

With the assistance of biomechanical modeling, a theory was proposed to determine how the supportive tissues of the bladder became defective during vaginal delivery. This theory proposed that during vaginal childbirth, descent of the fetal head to the level of the pericervical ring causes significant tensile and shear stress and strain on the tissue of the pubovervical fascia. As the birth canal narrows at the level of the ischial spines, the narrowest diameter of the pelvis, stress and strain are significantly concentrated because the tissue must undergo greater deformation in order to accommodate the fetal head. Internal rotation of the fetal head occurs in order to present the optimal diameter of the fetal head to the bony pelvis. This rotation of the fetal head induces transverse shearing forces onto the pubocervical fascia, already under high loading strain caused by the fetal descent. The strained and shearing forces can exceed the strength of the pubocervical fascia, resulting in soft tissue tears. The superior and inferior direction of the shear stress and strain caused by fetal descent and from the internal rotation of the fetal head causes soft tissue tears to occur in a transverse direction at the level of the pericervical ring.

While the pubocervical fascia undergoes significant tensile loading during fetal passage, extensible anisotropic materials, such as soft tissue, are more resistant to tensile stress and more likely to fail as a result of shear stress, as given by the criteria of maximum shear stress. This strongly suggest that the tears of the pubocervical fascia from the pericervical ring are more likely to occur as transverse tears of the pubocervical fascia from the pericervical ring rather than vertical (midline) or lateral (paravaginal tears) (Fig. 4.1).

The lateral levator arches lie up and out of harms way just inferior to the pelvic inlet, and these structures are less likely to deform, during vaginal birth or be injured during this process. This suggests that lateral (paravaginal) injury is less likely to suffer from the tensile stress to these tissues and less likely to fail or tear as a result of shear stress.

In the later half of the twentieth century, gynecologic surgeons thought it was possible to diagnose the types of vaginal prolapse by pelvic examination prior to surgical correction. Although this was an admirable goal, the conclusions of these observations were guided by only two options

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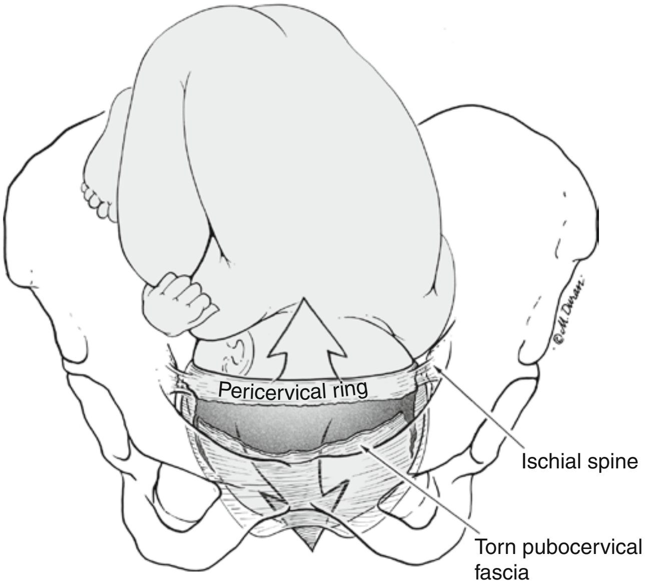
Pericervical ring
Ischial spine
Torn pubocervical fascia
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Fig. 4.1 Proposed transverse fascial tear of the pubocervical fascia from the pericervical ring during fetal descent and internal rotation of the fetal head

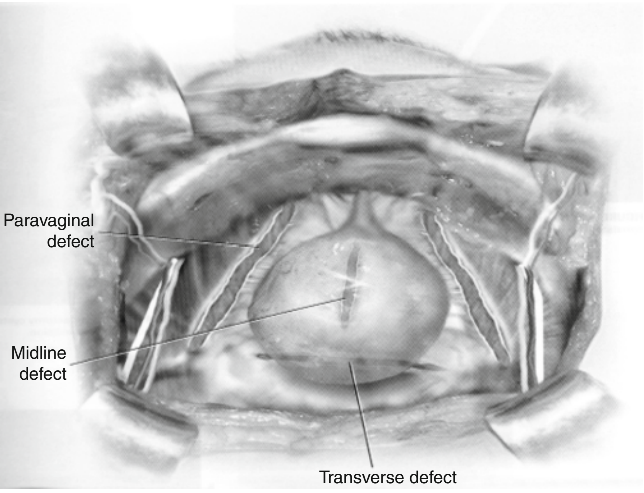
Paravaginal defect
Midline defect
Transverse defect

Fig. 4.2 Abdominal view of proposed defects in the connective support of the anterior segment of the pelvis (Modified from Richardson et al.[3])

thought to cause prolapsed of the anterior and posterior vaginal walls – midline or lateral tears.

Cystoceles and rectoceles were thought to be hernias. A hernia is a protrusion of part of a structure through tissues normally containing it. Thus, the bladder or rectum must protrude through the fascia normally containing it to be called a hernia. Defects have been well documented in the gynecological literature (Fig. 4.2), yet the herniation of the bladder through a midline or paravaginal defect, which were thought to be recognized occur during pelvic examination or with operative repairs, has never accurately been described or observed. More effort has been spent discussing various proposed vaginal defects than documenting the actual herniation of the bladder or rectum protruding through the structures that normally contain it (Figs. 4.3–4.5).

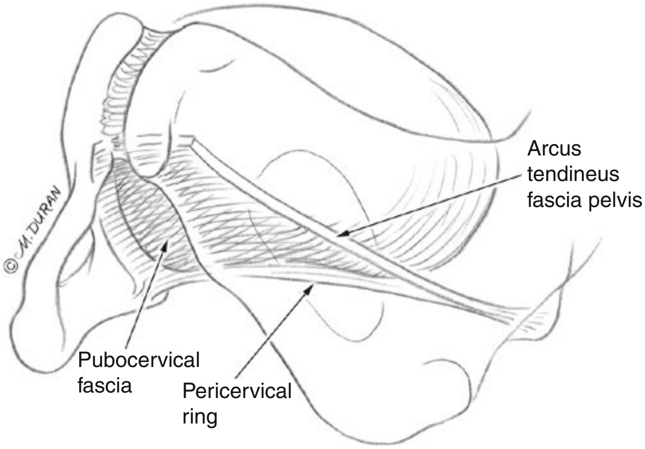
Arcus tendineus fascia pelvis
Pubocervical fascia
Pericervical ring
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Fig. 4.3 Normal support of the bladder by the supportive tissues (pubocervical fascia) of the pelvis

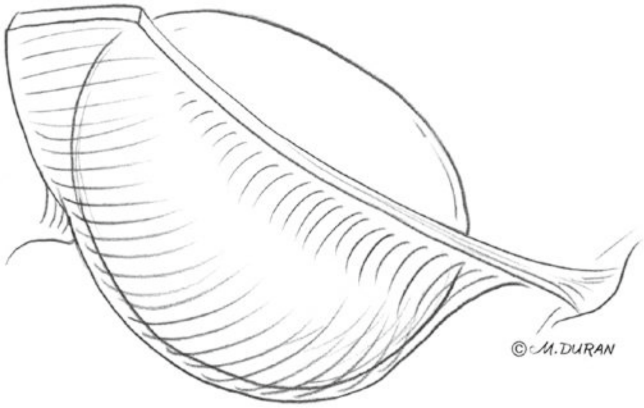

Fig. 4.4 Howard Kelly's proposed stretching of the pubocervical fascia causing anterior vaginal wall prolapsed

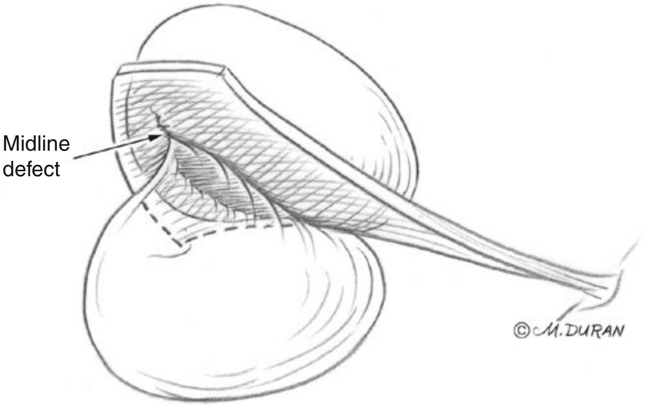
Midline defect
©M.DURAN

Fig. 4.5 Cullen Richardson's proposed midline tear and defect in the pubocervical fascia, with the bladder protruding through the midline tear of the pubocervical fascia

The best approach to anterior and posterior vaginal wall prolapse has long been debated. In my experience, most reparative failures are due to incorrect diagnosis of the defects, which leads to incorrect repair. Only when these defects are accurately identified and precisely corrected is anatomic cure possible.

The Baden Walker Hafway System[4] or the POP-Q[5] examination is recommended during physical examination. Kovac and Zimmerman[11] uses a pelvic organ prolapse map that is perhaps the most useful. The physical examination should be documented in a fashion that is accurate, reproducible, and complete. However, the question remains whether or not the anatomic cause of the herniated bladder or rectum can be accurately identified during pelvic examination. It is widely accepted that the presence of vaginal rugae indicates the presence of underlying supportive fascia. Baden and Walker offered their S-H-E straining test as a method to assess the severity of vaginal support loss. They suggested that the role of this test was to delineate the lines for transvaginal incisions and for the placement of transabdominal paravaginal sutures in the operating room. Paravaginal defects were for a while thought to be extremely common and believed could be detected by pelvic examination.

It was also believed loss of support along the entire vaginal canal could be accurately reflected by movement of the vaginal walls during valsalva. Baden and Walker[7] in 1992 proposed an anterior vaginal "window" defect caused by either a precervical fascial avulsion, midline defect, or probable paravaginal detachments (Fig. 4.6). They were almost on target when they considered the possible cause, a precervical fascial avulsion; however, they were probably influenced by the belief of the times by considering midline and paravaginal defects as the only possible causes. Observe the absence of vaginal rugae and the smooth vaginal epithelium, "window defect" depicted in Fig. 4.6. I have come to understand that these findings more likely represent a transverse defect, perhaps a precervical fascial avulsion (Fig. 4.7). Separation of the anterior pubocervical fascia from the pericervical ring best explains the smooth appearance of the vaginal epithelium and the absence of fascia, which has retracted superiorly (Fig. 4.7). Midline plication of the underlying bladder or reattaching the fascia to the vagina or white line cannot possibly correct the herniated bladder through the separated pubocervical fascia from the upper most part of the vagina or percervical ring. From Baden and Walker's conclusions, medial movement of the lateral grooves with a speculum within the vagina when the patient is asked to valsalva would be diagnostic of paravaginal defects (Fig. 4.8). However, this interpretation can be misleading as the anterior blade of the Graves speculum may be holding the bladder in the midline, displacing the bladder to both lateral sides of the speculum if the defect occurs transversely (Fig. 4.9).

Gynecologists also proposed that a midline defect may be demonstrated by physical examination. Baden and Walker used their defect analyzer to detect midline defects. They believed that placement and elevation of their analyzer blades at the lateral sulci allowed a central bulge of a midline defect to further protrude with valsalva. I believe the central bulge

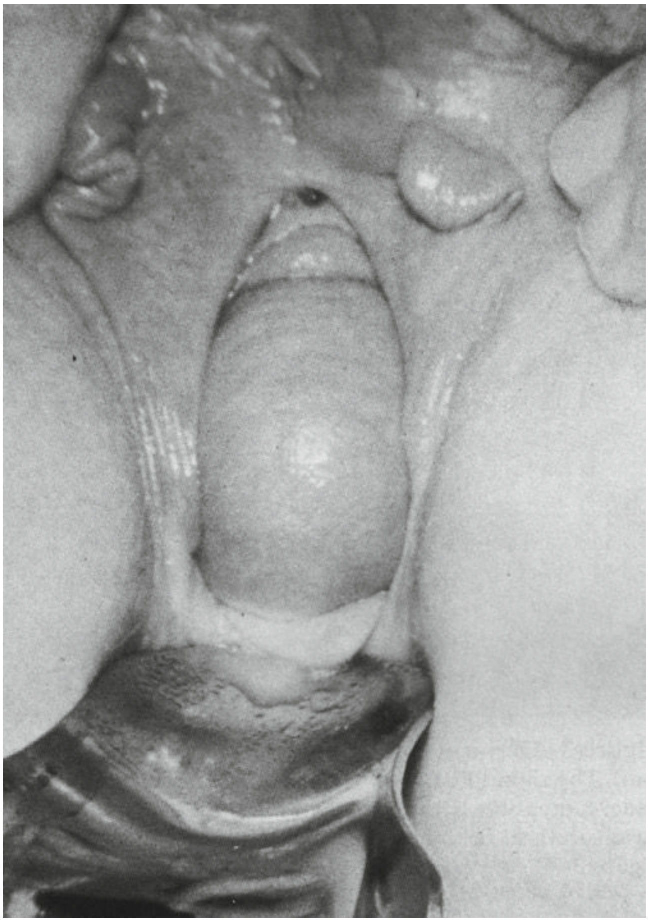

Fig. 4.6 Anterior vaginal window defect. Vaginal view of this Grade 3 cystocele, with smooth epithelium and extensive length of the anterior wall, is typical of a high fascial window caused by a precervical fascial avulsion, midline defect, and probable paravaginal detachment (Reprinted from Baden and Walker[7], copyright Elsevier 1992. With permission)

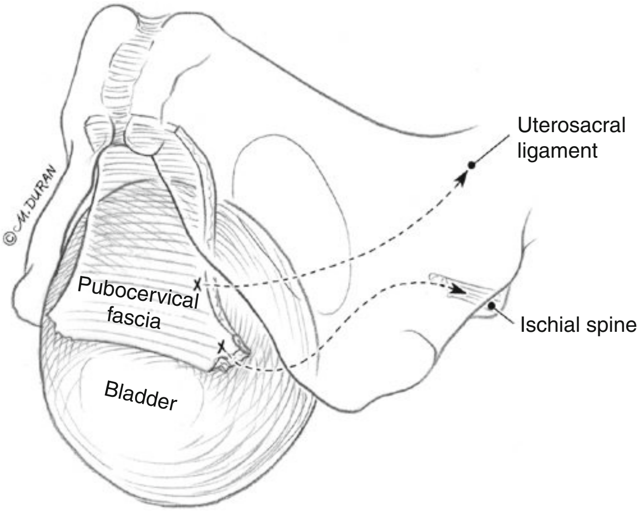

Fig. 4.7 Compare Figs. 4.6 and 4.7. Smooth epithelial appearance in Fig. 4.6 more likely represents the bladder in direct contact with the vaginal epithelium: as the pubocervical fascia is separated from the pericervical ring, it retracts superiorly. The vaginal rugae in Fig. 4.6 represent the intact portion of the pubocervical fascia seen in Fig. 4.7

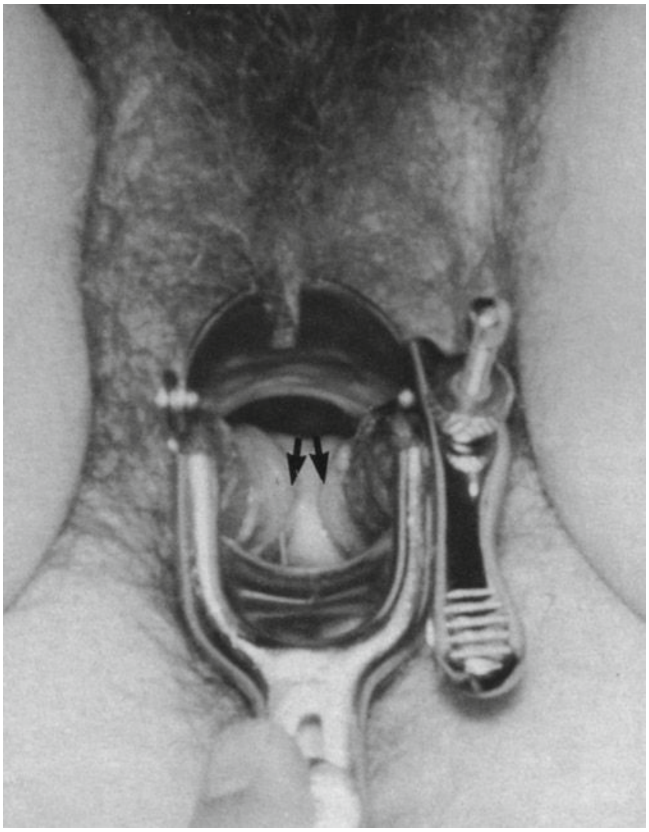

Fig. 4.8 Proposed bilateral paravaginal defects. When patient strains down firmly, vaginal grooves move medically to almost touch the midline (*arrows*) owing to severe loss of paravaginal attachments (Reprinted from Baden and Walker[7], copyright Elsevier 1992. With permission)

would more likely be from the herniated bladder through the separation of the pubocervical fascia and the pericervical ring in the midline (Fig. 4.10).

In 1976, Richardson and colleagues published their work of unilateral paravaginal fascial repair via the abdominal approach. This encouraged researchers and surgeons to believe that midline plication misses the real defect, as the real defect was thought to be lateral. Their repair attached the 2–3 cm of the vaginal sulcus, beginning at the bladder neck to the anterior tendinous arch to correct urinary incontinence. Subsequently, a significant number of ‘high” cystoceles recurred, so it was thought that the paravaginal sutures needed to be extended along the tendinous arch to the ischial spine. After Richardson et al.’s 1981[8] studies on paravaginal defect repair, it was suggested that traditional midline placation might be replaced by paravaginal repairs. This has not proven to be correct.

For the past 100 years, gynecologists were taught that repair of cystoceles and rectoceles were either performed by midline plications or paravaginal repairs. Does this mean injury to the supporting tissues during childbirth only occur as either vertical (midline) or lateral (paravaginal) tears? How the supporting tissues tear during vaginal birth in the midline or paravaginally has never been adequately explained. The hypothesis offered in this chapter from biomechanical modeling and our findings during surgery and cadaveric dissections suggest that transverse tears of the pubocervical fascia from the pericervical ring is the most likely cause of anterior vaginal wall defects (Fig. 4.10). The operative technique to correct the herniation of the bladder through the

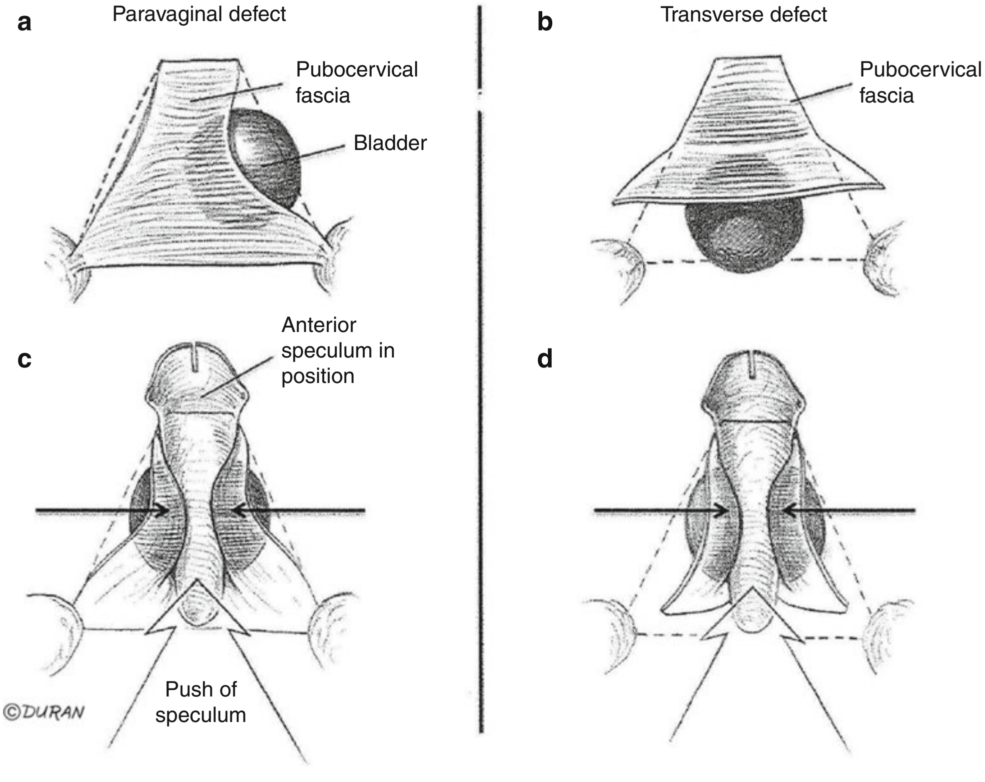

Fig. 4.9 (**a**) Paravaginal defect. (**b**) Transverse defect. (**c**) Placement of a speculum in the vagina more likely represents a transverse defect displacing the bladder to both lateral sides of speculum (**d**)

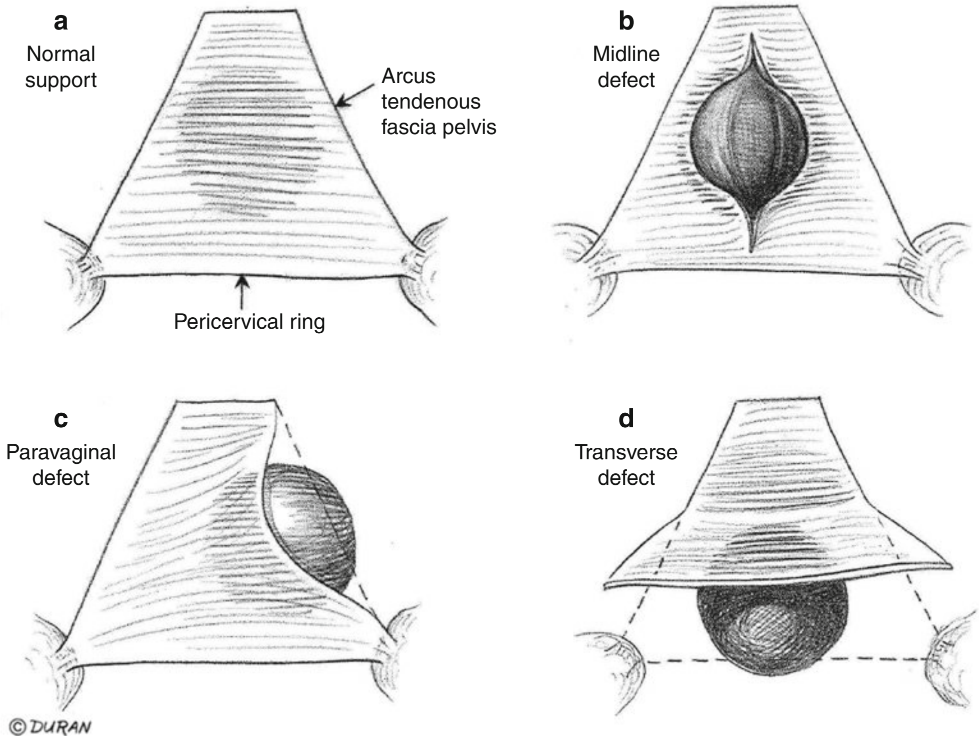

Fig. 4.10 Vaginal view of proposed defects of anterior vaginal wall prolapsed. (**a**) Represents the bladder supported by the trapezoidal pubocervical fascia. (**b**) Represents a proposed midline fascial tear with the bladder protruding through the proposed defect. (**c**) Represents the bladder protruding through a proposed paravaginal defect. (**d**) Represents a transverse defect with the bladder protruding through the defect caused by the separation of the pubocervical fascia from the pericervical ring

endopelvic fascia with a proposed midline defect has never described the bladder herniating through an actual tear or midline separation of the pubocervical fascia, and surgical descriptions have never described reducing the herniated bladder through such a defect before completion of the midline plication (Fig. 4.5). Similarly, description of an abdominal paravaginal defect repair does not describe reducing a herniated bladder, protruding out of the vagina through a paravaginal defect before the defect is repaired by suturing the fascia to the vagina or white line (Figs. 4.11 and 4.12).

Because prior to now no one has offered an alternative to midline and paravaginal defects to diagnose cystoceles and

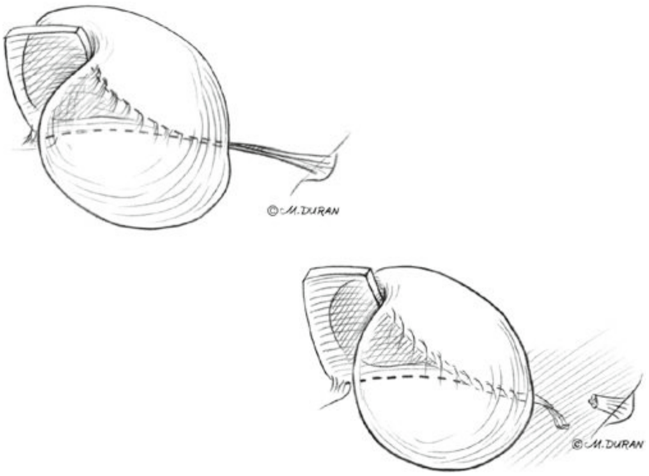

Fig. 4.11 Proposed paravaginal defect with bladder protruding around a proposed paravaginal defect and protruding out the vagina

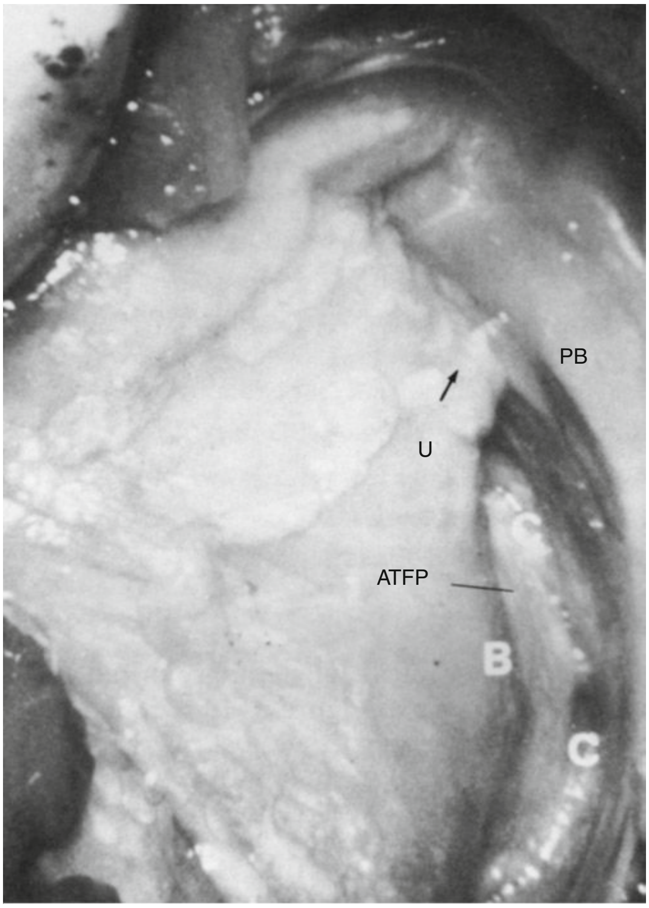

Fig. 4.12 Paravaginal defect (C) Posterolateral pubic bone (PB) lies at the *top right* corner. Bladder has been filled with 90 ml of urine. Note bladder edge (B) Does not protrude into paravaginal defect (Reprinted from Baden and Walker[7], copyright Elsevier 1992. With permission)

rectoceles, present attempts to correct midline or lateral defects must be seriously reevaluated. Any description of a vaginal defect based on the concept that they are either midline or paravaginal herniations without the herniation of the bladder or rectum through the structures designed to contain them are obliged to document the actual herniated structures. Similarly, general surgeons document hernias through the anterior abdominal wall hernias before they surgically correct these hernias.

DeLancey[9] conceptualized that each portion of the vagina relies on different levels of support to preserve normal vaginal anatomy. He proposed that the middle third of the vagina is supported by the lateral attachments of the pelvic sidewall at the arcus tendineus fasciae pelvis. It was suggested that any operation that does not account for attachment to the arcus for middle third-level defects (cystoceles and rectoceles) is likely to fail. If paravaginal defects are not the cause of anterior vaginal wall prolapse, then reattachment of the fascia to the lateral attachments (arcus) may not be appropriate and unnecessary.

The posterior segment of the vagina is also deformed by the effects of childbirth. As the fetus descends and passes under the pubic arch, considerable stress occurs to the posterior pericervical ring. This results in further stress on the uterosacral ligaments with a transverse proximal detachment of the rectovaginal septum at its junction with the pericervical ring (Fig. 4.13). This is the most common overall injury from fetal damage during vaginal delivery. As with the anterior segment of the vagina, most gynecologic and colorectal surgeons continue to perform midline plication or lateral (pararectal) repairs of the posterior segment with the same poor results. The transverse separation of the rectovaginal fascia from the pericervical ring and uterosacral ligaments appears to be the most common defect causing rectoceles. Since the pericervical ring is where the deep endopelvic connective tissue support structures converge, the reconstructive vaginal surgeon's goal is restitution of the anatomical connections of the pericervical ring for both cystoceles and rectoceles. Apical support seems to be the most important repair for both anterior and posterior reconstruction. DeLancey[9] noted that if the dissection and reconstructive efforts during surgery do not extend to the interspinous diameter, the surgery is likely to fail. I strongly believe that apical support must be superior to the interspinous diameter to the level of the uterosacral ligaments as they insert into S-2, S-3 of the sacrum.

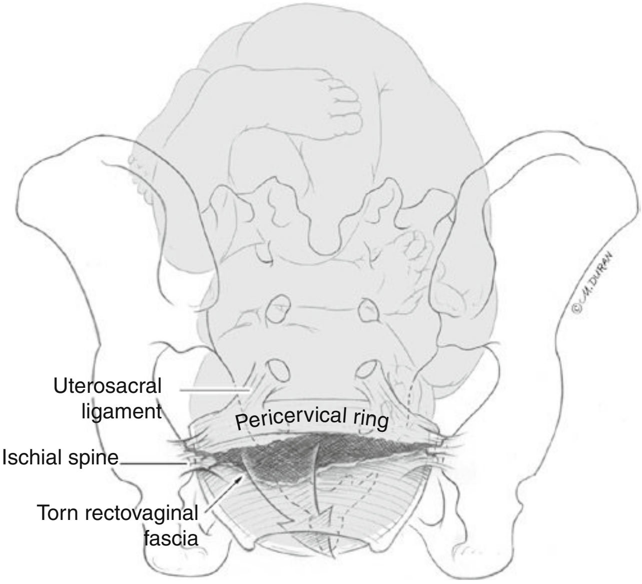

Fig. 4.13 Separation of the rectovaginal fascia from pericervical ring during vaginal delivery

Rectoceles have been considered to be a herniation of the rectum and posterior vaginal wall into the lumen of the vagina. Nichols[10] describes three types of damage: (1) stretching or attenuation of the full thickness of the vaginal wall after overdistension during childbirth; (2) stretching of the lateral attachments of the vagina to the pelvic sidewall, particularly the vaginal portion of the cardinal ligament; or (3) avulsion of the lateral attachments from the lower vaginal walls.

Nichols and others have also suggested that most rectoceles are also the result of separation of the pubocervical fascia from the perineal body. In my observation, reattachment of the fascia to the perineal body, inferiorly not apically, with midline plication has been the most common repair performed by gynecologic surgeons. This is also unpopular with patients because it has failed to resolve fecal splinting, which is the most common symptom that patients want resolved.

My operative experience and those observations of Zimmerman[12] have convinced me that most rectoceles are the result of separation of the rectovaginal fascia from the uterosacral ligaments and the posterior pericervical ring (Fig. 4.13). If cystoceles are the result of transverse tears of the pubocervical fascia from the anterior part of the pericervical ring, then are not rectoceles caused by transverse tears of the rectovaginal fascia from the uterosacral ligaments and the posterior pericervical ring during childbirth? (Fig. 4.14). It seems simple. Those who still support midline or lateral repairs of rectocele or cystocele must identify and document two issues: (1) how and in which direction these tears occur during vaginal birth and (2) can the rectum or bladder be identified herniating through a midline defect; or does the protrusion of the prolapsed vagina represent the bladder or rectum herniating through a paravaginal or pararectal defect. Apical reattachment of the rectovaginal fascia to the posterior percervical ring and to the uterosacral or sacrospinous ligaments is an operation for which many gynecologists finally respect (Fig. 4.14).

Correcting the separation of the pubocervical fascia from the pericervical ring and providing apical support to the iliococcygeal fascia inferior to the ischial spine as well

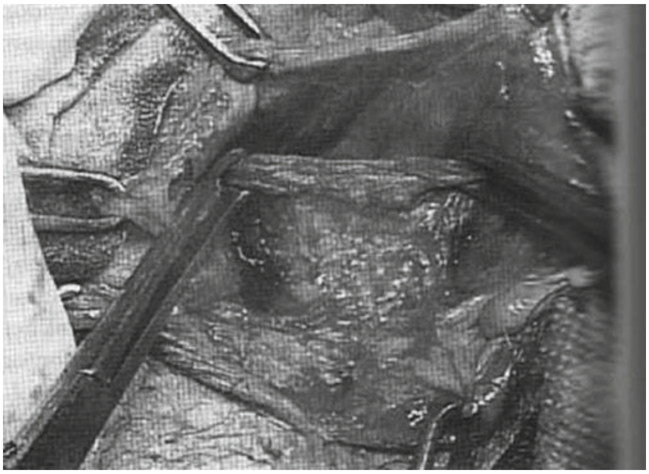

Fig. 4.14 Fully exposed rectovaginal septum detached from the uterosacral ligaments causing rectoceles. The edge of this septum must be attached apically to the posterior paracervical ring and to the uterosacral or sacrospinous ligament to properly correct a rectocele

as the retroperitoneal uterosacral ligaments has been tested in an IRB study at Emory University on 259 patients with Stage 3/4 anterior vaginal wall prolapsed. These patients were followed for 24 months. A success rate of 95% was achieved. Comparing these results to the 40–60% failure rates of midline plication or paravaginal repairs appear to suggest that this may be the most appropriate repair for prolapse of the anterior vaginal wall.[12] A multicenter study is currently under way to further test the validity of this new concept and repair.

My goal of this chapter was to introduce restitution of the anatomical connections of the supportive tissues of the bladder and rectum to the pericervical ring for both cystocele and rectocele repairs. I have challenged the concepts of midline and lateral repairs for which most of us have accepted. These beliefs from the understanding of surgical anatomy of 100 years ago for midline plication and 60 years ago for lateral (paravaginal) repairs need further evaluation. I hope this chapter will provide the stimulus to question the continued use of these old techniques with their dismal failure rates.

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Complimentary Investigations

5

Deborah R. Karp and G. Willy Davila

Investigation of the urogynecologic patient requires a comprehensive, patient-oriented approach, which involves a detailed patient history, a focused genitourinary examination, a neuromuscular assessment of the pelvic floor, and other necessary complementary studies. Urodynamic studies, cystoscopy, and imaging such as anorectal ultrasound and functional defecatory evaluation can add essential information to guide conservative and/or surgical treatment for the patient. Validated questionnaires allow for a standardized approach to assess patient symptomatology, quality of life, disease impact, and therapeutic effects following medical or surgical treatment.

Urogynecology History

Urinary and fecal incontinence and pelvic organ prolapse are common conditions in aging women. Stress urinary incontinence (SUI) is the loss of urine with activities associated with increases in intra-abdominal pressure, such as coughing, sneezing, laughing, and exercise. The rate of SUI in women peaks at the age of 45–49 reaching 65%.[1] Women with urge urinary incontinence typically complain of urine loss accompanied by a strong sense of urgency and often complain of nocturia and daytime urinary frequency. Urge incontinence is the result of spontaneous, involuntary detrusor contractions that occur without warning. The prevalence of detrusor overactivity is greatest at the extremes of age with a 5–10% prevalence in premenopausal women, 38% prevalence in elderly, and over 80% prevalence in institutionalized incontinent elderly patients.[2]

It is important for a patient with urinary leakage or voiding dysfunction to complete a voiding diary. Voiding diaries record daytime and nighttime urinary events, leakage events, symptoms associated with urinary leakage, and the amount and type of fluid intake (Fig. 5.1). They can be helpful in determining the cause, type, and severity of urinary symptoms. Voiding diaries also provide a record of patient symptoms as patient recall is often unreliable and inaccurate. For urinary incontinence, performing a 3-day diary is sufficient, as a 7-day diary has been associated with a lower degree of patient compliance.

There are many important variables that should be obtained via an accurate and comprehensive patient history. The use of daily pads to absorb urine is associated with more severe forms of urinary incontinence, such as intrinsic sphincter deficiency (ISD). Voiding dysfunction is common in the elderly incontinent patient population. Patients should be asked if they have difficulty initiating urination, interrupted, slow, or double voiding, incomplete emptying, whether they strain to urinate, or if they have frequent or recurrent urinary tract infections. Patients with pelvic organ prolapse typically complain of pelvic pressure, lower back pain, or a sensation of fullness. Symptoms often gradually worsen over time as the prolapse becomes more severe and manifest as a palpable, exteriorized lump with prolonged standing, strenuous activities, or at the end of the day. Other commonly associated conditions involve sexual dysfunction, fecal incontinence, and obstructed defecation. Because they are problems that cause social stigma and embarrassment, it is essential to gather information on the impact of patient symptomatology on social functioning and quality of life. This may require a quality of life questionnaire.

Conditions that cause chronic increases in intra-abdominal pressure such as obesity, chronic coughing, a history of smoking, and other pulmonary conditions predispose a patient to pelvic organ prolapse and urinary incontinence. Neurologic disorders including multiple sclerosis, cerebrovascular accidents, thoracolumbar vertebral disease, and Parkinson's disease may be the cause of urge incontinence and voiding dysfunction.

Previous pelvic surgeries such as hysterectomy, anti-incontinence procedures, and the use of graft materials have important treatment implications, because they alter the normal supportive structures of the pelvis. Previous pelvic

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VOIDING DIARY

Time	Urinations in toilet	Urge	Leakage accidents			Fluid intake 1 = Teacup 2 = Glass/can 3 = Water bottle
			Urge	Stress	Other	
12 midnight–2 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
2–4 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
4–6 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
6–7 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
7–8 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
8–9 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
9–10 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
10–11 a.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
11 a.m.–12 noon	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
12 noon–1 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
1–2 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
2–3 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
3–4 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
4–5 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
5–6 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
6–7 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
7–8 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
8–9 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
9–10 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
10–11 p.m.	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3
11 p.m.–12 midnight	☐ ☐ ☐ ☐	☐ ☐	☐	☐	☐	☐1 ☐2 ☐3

Fig. 5.1 (Voiding diary): The voiding diary is a 3-day log of the number of urinations, urgency episodes, leakage events, and associated urgency and/or stress symptoms, and the amount and type of fluid intake

radiation may lead to severe incontinence due to an alteration of the viscoelastic properties of the bladder and/or bladder neck. Multiple vaginal or operative vaginal deliveries, large obstetric tears, and macrosomia are important components of a complete obstetric history. A family history of incontinence and prolapse indicate predisposition due to alterations in collagen and elastin type, structure, and function.

Commonly prescribed drugs contribute to voiding difficulties, urgency and frequency, and incontinence. Antihistamine use can lead to urinary retention and voiding difficulty. Alpha-adrenergic agonists (such as phenylephrine) and antagonists (such as prazosin) have opposing direct effects on urethral sphincter function and lead to urinary retention or stress incontinence, respectively. Other drugs that may lead to urinary retention and voiding dysfunction include antidepressants, antipsychotics, and anticholinergic medications for overactive bladder (oxybutynin, tolterodine, solifenacin, etc.), because they cause relaxation of the detrusor muscle. Diuretics can worsen a patient's incontinence.

Lifestyle and dietary factors must also be assessed. Caffeine and alcohol intake worsen urinary urgency, frequency, and possibly urge incontinence. Likewise a patient's diet and dietary supplements should be analyzed to identify bladder irritants including citrus, spicy foods, and other high oxalate foods such as nuts, berries, chocolate and beets.

Current Validated Questionnaires: Urinary Incontinence and Pelvic Organ Prolapse

Pelvic floor pathology rarely results in severe medical morbidity or mortality; rather it affects quality of life. Because of this, there is an emphasis on quality of life impact assessment. Using validated questionnaires helps to precisely determine the impact of pelvic floor disorders on quality of life from the patient's perspective. Following treatment, questionnaires can be used to follow patient's progress, as objective measures such as prolapse stage and other objective parameters often do not correlate with patient perceived treatment outcome and goals.[3]

In general, questionnaires are divided into symptom impact or bother, quality of life, and sexual function questionnaires (Table 5.1).The Urogenital Distress Inventory (UDI) and its short form (UDI-6) are validated questionnaires that assess the degree to which a patient's incontinence

Table 5.1 Validated questionnaires for urinary incontinence and prolapse assessment

Abbreviation	Full name	Objective	Scale details
Symptom questionnaires			
UDI	Urogenital distress inventory	Assess degree to which lower urinary tract symptoms are bothersome	19 questions with 3 subscales (obstructive/discomfort, detrusor overactivity/irritative, stress incontinence)
UDI-6	Short version UDI		6 questions, correspond with urodynamic findings
PFDI	Pelvic floor distress inventory	Assess effect of pelvic floor disorders on quality of life	46 questions with 3 scales: urinary, pelvic organ prolapse, colorectal
PFDI-20	Short version		20 questions with 3 scales
Wexner CCF-FI	Wexner Cleveland Clinic Florid, Fecal Incontinence	Most commonly used fecal incontinence severity scale	5 questions with equal weight to solid, liquid, flatal incontinence, pad use, and lifestyle modifications
Quality of life questionnaires			
IIQ	Incontinence impact questionnaire	Assess life impact of urinary incontinence symptoms	30 questions with 4 domains (physical activity, social relationships, travel, emotional health)
IIQ-7	Short version IIQ		7 item short form
King's Health Questionnaire	King's Health Questionnaire	Assess quality of life with urinary incontinence	32 questions with 10 domains; 8 validated cultural adapatations available in 26 languages
PFIQ	Pelvic Floor Impact Questionnaire	Assess life impact of pelvic floor symptoms	93 questions with 3 scales: urinary, pelvic organ prolapse, anorectal
PFIQ-7	Short version PFIQ		7 questions with above 3 scales
FIQL	Fecal Incontinence QOL Scale	Assess life impact of anal incontinence	29 questions with 4 domains: lifestyle, coping/behavior, depression/self-perception, embarrassment
Sexual function questionnaires			
PISQ	Pelvic Organ Prolapse/ Urinary Incontinence Sexual Questionnaire	Assess sexual function in patients with urinary incontinence and/or pelvic organ prolapse	31 questions with 3 domains: behavioral/emotive, physical, and partner-related
PISQ-12	Short version PISQ		12 questions with above 3 domains
FSFI	Female Sexual Function Index	Assess female sexual function and quality of life	19 question with 6 domains (disorders of: desire, arousal, lubrication, orgasmic, satisfaction, pain)

symptoms are bothersome to her, and the Incontinence Impact Questionnaire (IIQ) and its short form (IIQ-7) assess the impact of urinary incontinence on a patient's daily activities, social roles, and emotional states.[4,5] The Pelvic Floor Distress Inventory (PFDI) and its short form (PFDI-20) provide a comprehensive evaluation of the effect of pelvic floor disorders on quality of life.[6] In addition to the UDI, questions regarding pelvic organ prolapse and defecatory dysfunction are included. The Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire (PISQ), its short form (PISQ-12) and Female Sexual Function Index (FSFI) are validated questionnaires pertaining to sexual function.[7,8] These questionnaires allow a standardized means to assess symptoms, quality of life, sexual function, and other subjective measures that may not necessarily correlate with objective data. Due to the frequent coexistence of pelvic floor symptoms and variable associated individual impact, we recently developed a validated global pelvic floor bother questionnaire to identify and assess the impact of various pelvic floor symptoms.[9]

Colorectal History

Because of the shared innervation and close anatomic proximity of the colorectal and genitourinary tract, complex defecatory disorders and fecal incontinence are common in patients with urinary incontinence and pelvic organ prolapse. Patients should be asked if they strain with defecation, are incontinent of flatus, liquid, or solid stool, or need to manually assist themselves with defecation. For patients with constipation, outlet obstruction due to trapping of stool within a rectal wall hernia (rectocele), colonic inertia, neoplasm, and pelvic floor dysynergia are a part of the differential diagnosis. Rectoceles may be symptomatic by the need for splinting or applying manual pressure within the vagina, rectum, or perineum. If rectal bleeding is present, occult malignancy should be ruled out with colonoscopy or barium enema.

Patients with a history of anorectal surgery such as hemorridectomy, anal sphincter laceration repair, and bowel resection are at risk for future anal incontinence. Traumatic vaginal delivery with large perineal lacerations, midline episiotomy, prolonged second stage of labor, operative vaginal delivery, and fetal macrosomia are additional risk factors for fecal incontinence. In addition, undiagnosed anal sphincter laceration following vaginal delivery is associated with the future development of incontinence of feces. Sultan et al.[10] reported clinically undetected anal sphincter tears in 35% of primiparous women 6 weeks after vaginal delivery.

Urge fecal incontinence, the unwanted loss of stool despite a voluntary attempt to inhibit defecation, signifies external anal sphincter damage. Passive soiling, or the passage of stool without the patient's awareness, may be indicative of internal anal sphincter pathology. The quality of stool provides a gauge of the severity of incontinence. Flatus is more difficult to control than liquid stool and liquid stool more difficult to control than solid stool.

There are several validated tools for the assessment of fecal incontinence before and following treatment. The Cleveland Clinic Florida Fecal Incontinence Score uses the frequency of incontinence to solid, liquid, and gas, and the frequency of protective pad use and lifestyle alterations to rate patient symptoms on a score from 0 to 20.[11]Zero is complete fecal continence, and 20 is complete fecal incontinence. Other scoring scales for fecal incontinence include the Fecal Incontinence Quality of Life (FIQL) questionnaire which scores 29 items to measure quality of life following medical or surgical treatment.[12] These validated questionnaires provide objective outcome measures to evaluate treatment efficacy.

Urogynecologic Physical Examination

A comprehensive urogynecologic evaluation involves a thorough lower extremity and perineal neurologic exam, abdominal, gynecologic, and rectal examination. Any evidence of abdominal or pelvic masses, or hernia defects should be noted.

Pelvic floor neurologic evaluation includes testing sacral reflexes which involve the bulbocavernosus and anal wink reflexes. The absence of reflexes or presence of asymmetric reflexes or hyperreflexia is suspicious of an underlying neurologic condition and warrants a full neurological evaluation.

It is important to evaluate all urogynecologic patients for signs of urogenital atrophy, especially if graft use is being considered for reconstruction. Symptoms suggestive of atrophic vaginitis do not necessarily correlate with physical signs of urogenital atrophy.[13] Objective measures are thus important for assessing and treating urogenital atrophy. Untreated hypoestrogenemia and atrophy may contribute to irritative urinary complaints such as urinary urgency and frequency, exacerbate symptoms of prolapse, and alter healing following vaginal surgery. Physical signs of urogenital atrophy include vaginal pallor, dryness, petechiae, and friability and the loss of vaginal rugations. A $pH > 4.5$ indicates either urogenital atrophy or bacterial infection. A vaginal maturation index, collected like a conventional pap smear, provides objective evidence of urogenital atrophy.

Other important exam findings are the presence of ulcerations along the vaginal walls, especially in women who are pessary users. A bimanual exam should also be done to rule out a pelvic mass, assess for pelvic pain and levator muscle tenderness, and quantify pelvic floor strength.

A comprehensive examination includes a visual assessment of the perineal body and rectal examination. One should look for rectal mucosal prolapse, scarring from previous episiotomy or laceration repair, or a wide open or patulous anal sphincter. A digital rectal exam should be performed with close attention to sphincter tone both at rest and with voluntary contraction. The internal anal sphincter is responsible for 50–85% of the resting tone of the anal canal while the strength of rectal squeeze indicates external anal sphincter function. Fecal impaction is strongly associated with generalized pelvic floor dysfunction and concomitant voiding dysfunction.

Pelvic Organ Prolapse Evaluation

Pelvic organ prolapse must be evaluated in a systematic, site-specific fashion. The patient performs a strong Valsalva effort or coughs to demonstrate the full degree of descent of each specific pelvic organ. Using a standard vaginal Grave's speculum to isolate the cervix or vaginal apex, the presence of uterine and vault support is usually evaluated first. The speculum is then taken apart, and its posterior valve is used to isolate the anterior and posterior compartments separately. If a patient's physical findings do not correlate to her complaints or severity of symptoms, the patient should be examined in the standing position. For a patient with a vaginal pessary, it may be necessary to leave the pessary out and reexamine the patient at a later date in order to confirm the extent of prolapse.

The use of a standardized classification system to describe pelvic organ prolapse improves communication in clinical practice and research protocols and provides an objective method for surgical planning and outcome. There are two systems currently in place to quantify pelvic organ prolapse. The Baden-Walker Halfway system is a simple system in which each vaginal compartment (cystocele, enterocele, rectocele, uterus, and vaginal vault) is isolated, and the most dependent portion is assessed during maximal straining in relation to the hymenal ring and mid-vaginal plane. The second classification system is the Pelvic Organ Prolapse Quantification System (POP-Q).[14] The POP-Q system was developed and adopted by the International Continence Society in 1995. In this system, nine points are used to make site-specific measurements (Fig. 5.2). Measurements are then recorded on a 3 × 3 grid.

It is critical to assess and quantify pelvic floor muscular strength, because pelvic floor weakness plays an important role in the development and progression of pelvic floor dysfunction. During digital vaginal exam, palpating the levator ani muscle complex along the posterior vaginal wall while the patient is asked to "squeeze" around the examiner's finger will provide a measure of strength and duration of contraction and perineal lift. If there is an assymetric contraction, this should be documented.

Different grading scales for pelvic floor muscle strength exist. The Modified Oxford muscle-grading scheme used for skeletal muscle ranks muscle strength on a scale of 0–5 (Table 5.2).[15] Other grading scales for skeletal muscle strength involve subjective categorization of the strength of the squeeze as "absent," "weak," "moderate," or "good" and duration as "absent," "brief," or "normal."

Table 5.2 Grading scales of muscle strength: the Modified Oxford muscle grading scheme

Grading scale for levator ani strength	
Score	Levator Ani
0/5	No contraction
1/5	Flicker, barely perceptible
2/5	Weak, no discernible lift
3/5	Moderate, lifting of the muscle and elevation of the posterior vaginal wall
4/5	Good, pulls fingers in and up loosely, elevation of the posterior wall against resistance
5/5	Strong, pulls in and up snugly, strong resistance applied to the elevation of the posterior vaginal wall

From Laycock [15]. With permission from Elsevier

The Colpexin Pull Test provides a direct and objective measurement of pelvic floor strength[16] (Fig. 5.3). This test correlates well with the digital pelvic exam scale, and it has recently been validated as a reliable and objective measure that provides consistent and reproducible results.[17] To perform the pull test, a lubricated Colpexin sphere is inserted in the vagina above the levator ani complex with the patient in the dorsal lithotomy position. The sphere is attached to a handheld tensiometer and slowly withdrawn while the patient performs a pelvic floor contraction. The maximum force generated is recorded in pounds to three significant digits. Previously published literature has shown that a mean pelvic floor muscle contraction generates 2.27 ± 1.45 lb strength.[16]

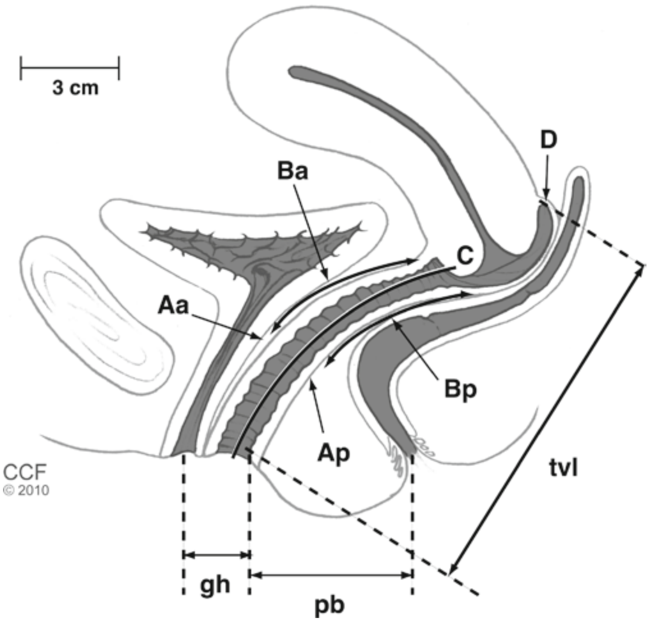

Fig. 5.2 The pelvic organ prolapse quantification system: nine individual points are used to assess pelvic floor descent (Courtesy of Ross Papalardo, Cleveland Clinic). Points Aa and Ba (anterior vaginal wall); Points Ap and Ba (posterior vaginal wall); Points C and D (vaginal apex); Points gh (genital hiatus); Points pb (perineal body)

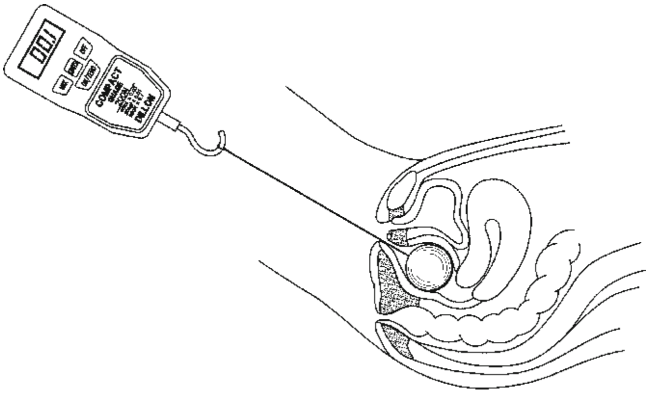

Fig. 5.3 The colpexin pull test: a new valid and reliable objective measurement of pelvic floor strength. A lubricated Colpexin sphere is inserted in the vagina above the levator ani complex with the patient in the dorsal lithotomy position, attached to a tensiometer, and slowly withdrawn while the patient performs a pelvic floor contraction

Urethral Sphincteric Function Evaluation

Urethral sphincteric function and anatomy should be evaluated in patients with stress incontinence or suspected occult incontinence associated with advanced prolapse. An empty supine stress test (ESST) is done immediately after the patient voids. The patient is asked to Valsalva, and any visible urine leakage indicates a positive test and possible ISD.[18] Palpation of the anterior vaginal wall and urethra may illicit urethral discharge or tenderness indicative of a urethral diverticulum or inflammatory condition of the urethra.

The Q-tip test is the most commonly performed test to measure mobility of the urethra. It is done by placing a sterile, lubricated cotton swab transurethrally to the level of the bladder neck with the patient in a horizontal supine position. The angle of the Q-tip in relation to the horizontal plane is measured with a goniometer or protractor at rest and with maximum Valsalva. Urethral hypermobility is defined as an angle of deflection of 30° or more with maximal Valsalva effort. Urethral hypermobility alone is not diagnostic of stress urinary incontinence, but is commonly used as a part of its treatment decision-making algorithm (Table 5.3).

Radiographic studies useful in the assessment of urethral anatomy involve cystourethrography, pelvic MRI and ultrasonography. In cystourethrography with contrast, lateral images taken at rest and with straining can demonstrate mobility or fixation of the bladder neck. Bladder neck funneling can be demonstrated signifying sphincteric compromise. The voiding component of cystourethrography can help visualize a urethral diverticulum, vesicovaginal or urethrovaginal fistula, bladder outlet obstruction or vesicoureteral reflux. Pressure cystourethrography with a Trattner catheter can diagnose urethral diverticulum; however, MRI imaging is considered the gold standard technique for the diagnosis of urethral diverticulum. Transperineal and introital ultrasound is another technique to assess the anatomy and function of the bladder base and urethra without the use of radiation and contrast materials and will be discussed in detail later in this chapter.

Table 5.3 Treatment algorithm of SUI

		Urethral function (UPP, VLPP)	
		Normal	Low
Urethral mobility (Q-tip test)	>30	Kegels, Physio, Pessary, TVT, Burch, TOT	Sling (traditional), ± TVT
	<30	Kegels, Physio	Bulking agents

TVT tension-free vaginal tape, *TOT* transobturator tape, *Physio* pelvic floor rehabilitation

Office Evaluation of Urinary Function

There are several simple office tests for urinary incontinence. For patients with incontinence, reversible forms of incontinence should first be ruled out and treated. These include infection, atrophy, drug side effects, metabolic causes, excessive fluid intake, restricted mobility, and impaired cognition. A baseline urinalysis to look for infection and hematuria is mandatory. In patients with persistent hematuria, urine cytology should be obtained from a voided specimen.

Uroflowmetry and Urinary Retention

Simple uroflowmetry and post-void residual can be performed in most gynecology and urology offices. The test begins by having the patient void with a full bladder in the most natural way possible. The time to void, flow time, and voided volume are recorded, and a post-void residual (PVR) is then immediately performed. Standardized normal values for a PVR have not been established. Volumes below 50 mL indicate normal bladder emptying and greater than 200 mL signify incomplete emptying and warrant further testing. Generally, a PVR of between 0 and 100 mL is considered to be normal.[19]

Simple Cystometry

With the catheter still in place and the patient in a sitting position, a syringe is attached to the end of the catheter, and the bladder is slowly filled with sterile water at room temperature. An acceptable bladder-filling rate is approximately 50–60 mL/min. Filling the bladder too quickly or with cold water can provoke spontaneous detrusor contractions in otherwise normal women. Care is taken to observe the water level in the syringe throughout bladder filling. Any spontaneous rise in the water level not associated with Valsalva, coughing, laughing, or movement may be due to a detrusor contraction. True bladder contractions are often accompanied by a sense of urgency and visible leakage of urine.

During bladder filling, the patient's first bladder sensation and maximum bladder capacity are noted. A normal bladder capacity varies from 300 to 600 mL. Once the bladder is filled to its cystometric capacity, the catheter is removed, and the patient is asked to cough. In order to mimic everyday stresses on the bladder, provocative measures (such as walking, jumping, running water, and heel bouncing) can be performed. Loss of urine associated with cough or Valsalva is diagnostic of stress incontinence. Any large amount of urine loss not associated with cough may indicate detrusor overactivity.

Another simple way to objectively demonstrate urinary incontinence is with the perineal pad test. This test can either be done in the office over 1 h or at home over a 24 h period. If done in the office, the patient's bladder is filled with 250 mL of sterile water, and the patient performs a standardized set of activities that typically affect incontinence (bending, coughing, straining, walking up stairs, or squatting) while wearing a pre-weighed pad. Any increase in weight of the pad represents the volume of urine loss. If there is uncertainty about urine loss, an agent such as phenazopyridine hydrochloride (Pyridium), which colors the urine, may make urine loss on the pad more obvious. The 24-h test is not as standardized as the 1-h test. False positive results may also occur due to sweat or vaginal discharge, and this test does not differentiate incontinence type.

Table 5.4 Complex urogynecologic issues requiring further evaluation

Urodynamic testing	Cystoscopy
• Uncertain diagnosis and treatment strategy	• Hematuria without infection
• Failed medical therapy	• Persistent urgency and frequency
• Consideration of surgical intervention	• Bladder pain
• Voiding dysfunction or elevated PVR	• Recurrent cystitis
• Exteriorized pelvic organ prolapse	• Suspected urethral diverticulum or fistula
• History of previous anti-incontinence surgery, pelvic radiation, or radical pelvic surgery	• Suspected foreign body, calculi, or iatrogenic bladder trauma
• Neurologic conditions such as multiple sclerosis or spinal cord lesions of injury	• When urodynamics fails to duplicate symptoms of urinary incontinence

Electronic Multichannel Urodynamic Testing

Urodynamic studies comprise a series of exams that provide a physiologic explanation of lower urinary tract symptoms. Testing apparatuses for urodynamics range from simple single channel methods performed manually, as described above, to more complex methods combining electronic measurements of bladder, abdominal, and urethral pressure with electromyography and fluoroscopy. Many different types of catheters including water-filled, air-charged and microtransducer catheters have been used. Microtransducer and air-charged catheters yield comparable results when evaluating urethral function with Urethral Pressure Profile (UPP) and Leak Point Pressure (LPP).[20]. Cystometry measures the pressure and volume relationship of the bladder and provides information on detrusor motor and sensory function, capacity and compliance. Uroflowmetry is a measurement of urine flow rate over time and is the initial screening test when voiding dysfunction is suspected. More complex urodynamic testing is indicated for cases of incontinence involving mixed symptoms, suspected fistula, urethral diverticulum, incomplete bladder emptying, exteriorized pelvic organ prolapse, failed treatments and therapy, and concomitant neurologic disease (Table 5.4).

Besides office manual cystometry described above, commercially available electronic cystometers are classified into single channel or multichannel units. Single channel cystometry measures intravesical pressure (Pves) during bladder filling. However, single channel measurements cannot determine whether a rise in vesical pressure is from a detrusor contraction or an increase in abdominal pressure. Multichannel cystometry provides a measurement of both the abdominal (Pabd) and intravesical pressures (Pves). The detrusor pressure (Pdet) is the difference between the intravesical and intra-abdominal pressure. Thus, Pdet = Pves − Pabd. In female patients, the abdominal pressure is obtained by placing a catheter transrectally or transvaginally. Transvaginal placement avoids artifact from rectal contractions. Transrectal placement is used in cases of advanced vaginal prolapse where vaginal catheters lead to inaccurate measurements.

In multichannel cystometry, urethral pressure can be measured to evaluate sphincteric function. A urethral sensor provides a measure of urethral pressure and allows calculation of maximum urethral closure pressure (MUCP) and functional urethral length (FUL). The MUCP is the maximum difference between the urethral and intravesical pressure. This test requires the use of a motorized puller arm to pull the urethral sensor along the length of the urethra. Although MUCP tends to be lower in women with genuine stress incontinence, there is an overlap between incontinent and continent patients. The MUCP is a passive assessment of instrinsic sphincteric function, mucosal coaptation, and extrinsic compression.

Whereas MUCP is a passive measurement, LPP measurement is an active assessment of urethral sphincteric function. The LPP is the peak bladder or abdominal pressure during Valsalva at which urinary leakage occurs. Although LPPs have been described in reference to both the bladder and abdomen, the detrusor leak point pressure (DLPP) has historically been used as a prognostic test to identify the risk of upper urinary tract deterioration in myelodysplastic children.[21] The abdominal leak point pressure is the intra-abdominal pressure at which leakage occurs in the absence of a detrusor contraction and quantifies stress incontinence by providing a numerical measurement of the resistance of the urethral sphincter to leakage. Leakage of urine with an abdominal pressure of less than 60 cmH_2O is suggestive of ISD.[22]

Various techniques have been developed to evaluate a patient's voiding mechanism. The simplest technique is uroflowmetry. It is merely a measure of voided urine volume over time. A uroflowmetric curve is a visual representation of urine flow over time to visually demonstrate voiding abnormalities (interrupted, prolonged, low flow patterns). Abnormal uroflowmetric results can occur from alterations in detrusor contractility, urethral resistance or both. Because uroflowmetry does not provide clear-cut information on bladder contractility or outlet resistance, it is best considered a screening test.

A pressure flow study should be performed to assess voiding function since abdominal, detrusor, and urethral pressures are simultaneously monitored. This test is most helpful at differentiating outflow obstruction from poor bladder contractility (underactive detrusor). An underactive or atonic detrusor muscle signifies bladder acontractility or areflexia due to a neurologic abnormality or denervation. Increased detrusor pressure on the other hand is associated with bladder outlet obstruction. Pressure voiding studies can also be helpful in evaluating denovo voiding dysfunction following slings or retropubic urethropexy, both of which may result in reduced urinary flow rates. Preoperatively, pressure-voiding studies provide valuable prognostic information on the likelihood of voiding difficulties following pelvic reconstructive and incontinence surgery.

When neurogenic voiding dysfunction is suspected, electromyography (EMG) is performed to assess urethral and anal sphincter activity. EMG records neuromuscular activity of striated muscles using an electrode either inserted into or placed onto the surface of a muscle. EMG surface electrodes qualitatively record overall neuromuscular activity. If desired, needle electrodes can be used to quantify the voluntary portion of urethral sphincter activity. Normally EMG activity should decrease and detrusor activity increase during voiding. The opposite should occur during filling. During urodynamics, the primary purpose of EMG is to detect detrusor sphincter dysynergia, a condition in which urethral sphincter contraction occurs during voiding leading to prolonged or interrupted voiding patterns and urinary retention. In defecation, EMG is used to diagnose anismus, in which levator ani contraction occurs with defecation resulting in obstructed defecation.

Videourodynamics combine urodynamic studies with fluoroscopic or ultrasound guided imagery of the lower urinary tract. Real-time images are taken during bladder filling, voiding, coughing, and straining. Bladder diverticulum, trabeculations, strictures, bladder neck funneling, and vesicoureteral reflux may be demonstrated. In cases of bladder outlet obstruction, the site of obstruction can usually be seen. The major disadvantages of videourodynamics include cost, radiation exposure to the patient and technician, and the technical expertise necessary to maintain its use. The use of videourodynamics should be limited to incontinent patients with neurological conditions, patients failing initial therapy, with bladder outlet obstruction, or in whom simple urodynamics do not lead to a definitive diagnosis. It is not indicated for routine evaluation of the urinary tract.

Neurophysiologic Testing

Neurophysiologic testing should be performed when the clinical evaluation of a patient with pelvic floor disorders suggests an underlying neurologic disorder. The most common tests used for evaluation of peripheral neurologic disease are EMG, nerve conduction studies, and sacral nerve reflex testing. Other testing may be required when central nervous system disease is suspected or present.

The primary nerve conduction study to assess pelvic floor function is the pudendal nerve terminal motor latency (PNTML). Nerve injury results in impairment of a nerve's conduction velocity leading to a delayed motor or sensory response or a prolonged latency. For PNTML, a disposable electrode attached to a glove, known as a St. Mark's electrode, is inserted into the anal canal. A stimulus is applied to the pudendal nerve at the level of the ischial spine, and its response is measured as a contraction of the external anal sphincter. Though each electrophysiology laboratory has its own established normal values, generally a PNTML greater than 2.0–2.4 ms is considered to be prolonged. Prolonged PNTML is indicative of pudendal nerve damage and has been associated with suboptimal results following treatment of fecal incontinence with sphincteroplasty.

Endoscopy of the Lower Urinary Tract

Although urodynamic testing provides an objective assessment of lower urinary tract function, it provides little information on urogenital anatomy. Cystourethroscopy is beneficial as a complementary test of the lower urinary tract, because it supplies information on anatomic abnormalities that contribute to lower urinary tract symptoms. Cystourethroscopy is indicated when a patient's symptoms suggest urethral diverticula, urogenital fistula, intravesical foreign body, interstitial cystitis, or bladder calculi or tumors. It is essential for the evaluation of hematuria and persistent irritative lower tract symptoms and in patients with continued incontinence following anti-incontinence and gynecologic surgery. Routine cystourethroscopy is not indicated for the work-up of uncomplicated incontinence in women.[23] It should not be performed in the setting of an active bladder infection.

Generally when performed for diagnostic purposes, cystourethroscopy is performed in the office. When performed in the operating room, it is used to evaluate ureteral and bladder integrity following pelvic reconstructive and incontinence surgery, facilitate surgical repair of urogenital fistula and diverticulum, assist in the safe placement of suprapubic catheters, perform bladder hydrodistension for the diagnosis of interstitial cystitis, aid in the biopsy of bladder lesions, and remove foreign bodies, bladder calculi or intravesical sutures.

Cystoscopic evaluation of the urethra may reveal polyps, foreign bodies, and diverticula. Anatomic assessment of urethral mucosal coaptation can be performed as a visual gauge of instrinsic sphincter deficiency or immediately following injection of periurethral bulking agents. The bladder mucosa normally has a smooth glistening surface. Any exophytic mucosal irregular lesions should be biopsied to rule out bladder cancer. Cystitis generally appears as hypervascularity, mucosal edema, and varying amounts of erythematous papular lesions. Vesicovaginal fistula following hysterectomy is typically located at the bladder base cephalad to the interureteric ridge corresponding to the level of the vaginal cuff and can vary from a few millimeters to a few centimeters in size. Cystoscopic findings of interstitial cystitis include petechial hemorrhages, linear cracking, significant glomerulations, Hunner ulcers, and terminal hematuria. Squamous metaplasia in the trigone area is a common normal finding in women with stress incontinence and appears as a thickened white cobblestone membrane.

Imaging of the Pelvic Floor

The prolapse exam is limited in its ability to accurately characterize the site and degree of prolapse due to variations in patient positioning, Valsalva strength, and prolapse of adjacent segments. Thus, radiographic studies have an increasingly important role in the accurate diagnosis and staging of pelvic floor prolapse. The advantages of imaging have yet to be determined, but imaging of complex pelvic floor pathology may improve our understanding of pelvic floor support mechanisms and prove useful in surgical planning. One study found that the addition of dynamic MRI changed the surgical plan in approximately 30% of cases, most often because of an occult enterocele not detected on physical examination.[24] Currently, the lack of standardization, validation, and universal availability of pelvic floor imaging modalities prohibit its use as widely adopted technique for the evaluation of pelvic floor problems.

Ultrasonography

Ultrasound is a widely used imaging modality in all pelvic floor disciplines. The advantage of ultrasound over conventional radiography for pelvic imaging is the lack of radiation exposure to the patient and the wide availability of ultrasound equipment. Three-dimensional ultrasonography is a new and emerging technology that provides the simultaneous visualization of pelvic organs in axial, transverse, and coronal views. Dynamic imaging allows anatomic assessment associated with movement (Valsalva and levator squeeze).

Ultrasound evaluation of the lower urinary tract and pelvis has been described from the transabdominal, transvaginal, transrectal, and transperineal (or translabial) approaches. Because of the significant shadowing generated by the pubic symphysis, the transabdominal approach is generally limited to bladder volume and post-void residual calculation and visualization of the renal pelvis and abdominal organs.

Transperineal three-dimensional ultrasonography offers dynamic functional and anatomic imaging. The probe is positioned on the perineum in the midsagittal plane and the pubic symphysis, urethra, bladder neck, vagina, cervix, and rectum are visualized (Fig. 5.4). The urethra is seen as a tubular structure with central echolucency and surrounding echogenicity due to its circumferential musculature. In the absence of significant pathology, the bladder will appear uniformly echolucent. Bladder wall contour can be assessed and wall thickness measured. Bladder wall thickening greater than 6mm is abnormal and may indicate infection, outlet obstruction, neoplasm, detrusor overactivity, or previous pelvic radiation.

The transperineal (or translabial) approach has become a popular method to assess prolapse because the probe does not enter the vagina and thus avoids unnecessary compression and artifactual distortion of anatomy. Though three-dimensional ultrasonography is not used routinely for the diagnosis and characterization of pelvic organ prolapse and urinary incontinence, anatomic alterations associated with

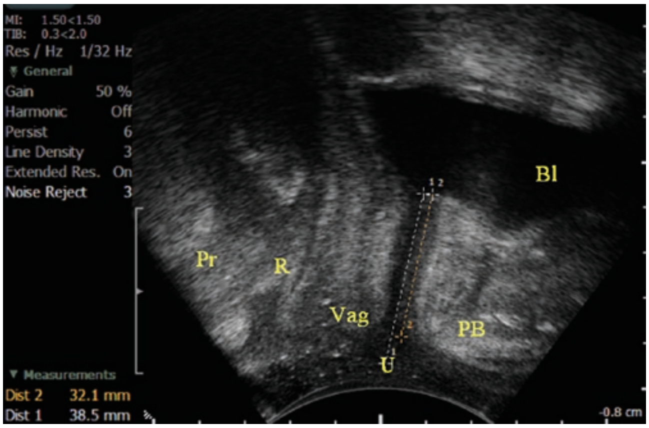

Fig. 5.4 Transperineal ultrasonography of pelvic floor: Dynamic imaging can be performed with Valsalva and squeeze. Important landmarks are: *PB* (pubic bone), *Bl* (bladder), *U* (urethra) with and without squeeze, *Vag* (vagina), *R* (rectum), *PR* (puborectalis)

these disorders has been characterized using this imaging modality. Enteroceles, known to be more difficult to identify on physical exam, can be easily seen by perineal ultrasound. In addition, quantification and staging of pelvic organ prolapse by transperineal ultrasound has been well correlated with POP-Q measurements by clinical exam.[25]

Another application of ultrasonography for lower urinary tract dysfunction is in the diagnosis and management of postoperative voiding dysfunction. The tension-free vaginal tape (TVT) has become a popular surgical treatment for female stress urinary incontinence. The TVT device uses a polypropylene tape to provide posterior support to the urethra by stabilizing the mid-urethra with increases in intra-abdominal pressure. On ultrasound, the TVT is echodense and easily seen posterior to the urethra (Fig. 5.5). In cases of postoperative bladder outlet obstruction, sharp angulation of the mid-urethra is seen at rest and severe narrowing of the urethral lumen suggests an over-tensioned sling.

Polypropylene implant materials have become very popular for the treatment of incontinence and pelvic organ prolapse over the past several years. The mediosagittal three-dimensional scan is best used to evaluate mesh width, thickness, configuration, and topographic position. Variants in mesh placement such as asymmetry, curling, bunching, and detachment can be seen as macroporous monofilament implant material is highly visible with ultrasound. Ultrasound has been shown to be superior for evaluation of periurethral tape position while MRI is more effective to depict the tape in the retropubic space.[26] Sonographic evaluation of polypropylene mesh after transvaginal prolapse repair has shown a significant discrepancy between size of the mesh at implantation and and postoperative mesh size. The reason for mesh contraction is not entirely known but may be explained by in vivo biomechanical or postoperative forces acting on the mesh causing distal retraction.[27] It is still not known if imaging assessment will be beneficial for surgical complications such as sling failure, denovo urgency, mesh erosion, and denovo dyspareunia. Research is needed to determine if mesh position and character will correlate with outcome.

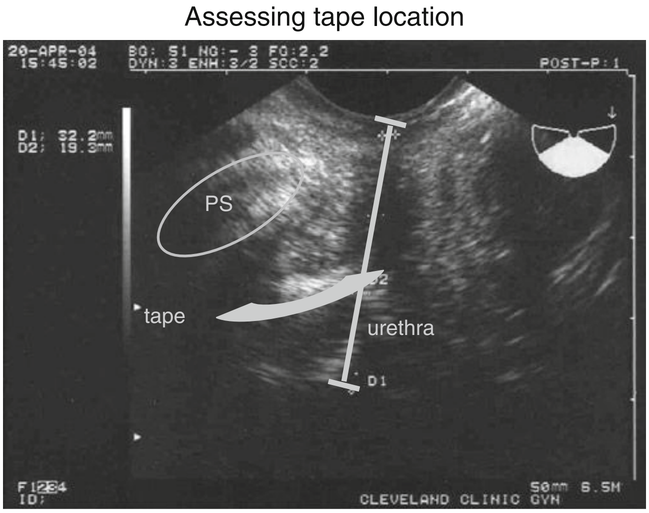

Fig. 5.5 Perineal ultrasound of TVT sling in place beneath mid-urethra: PS (pubic symphysis), tape (TVT sling)

Major levator trauma, that is, avulsion of the puborectalis-pubococcygeus from the pelvic sidewall can be seen using either magnetic resonance imaging or three-dimensional pelvic floor ultrasound. Levator trauma is generally a consequence of vaginal childbirth, is associated with vaginal prolapse, and has a prevalence of 15–35% in vaginally parous women.[28] The levator musculature is a highly echogenic complex on both translabial and introital three dimensional ultrasound. An avulsion injury is diagnosed if the muscle is seen detached from its insertion point on one or both sides. Inspection of the muscle during squeeze and with Valsalva increases the likelihood of detection of abnormalities. Avulsion of the puborectalis muscle is thought to be a risk factor for recurrent prolapse after surgical repair and is well associated with anal incontinence.[29] Surgical restoration of normal levator anatomy has recently been associated with restoration of normal pelvic floor function.[30]

Transrectal or endoanal ultrasonography is an excellent modality for the work-up of fecal incontinence and evaluation of the anal canal. The internal anal sphincter appears as a hypoechoic circular band; the external anal sphincter is seen as a thicker echogenic band just exterior to the hypoechoic internal sphincter muscle (Fig. 5.6). Defects to the musculature are seen ultrasonographically as breaks in the continuity of the sphincter ring. One can measure the extent of the defects in relation to the 360° view provided by the endoanal probe.

Endoanal ultrasound can also be used to identify rectovaginal fistulas. Fistula tracts typically have a hypoechoic appearance making them difficult to identify on ultrasound. The use of hydrogen peroxide transforms the tract to a more hyperechoic appearance due to its bubble producing properties and allows easier identification of the fistulous tract.[31]

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) has the ability to noninvasively survey the pelvis. It allows the precise differentiation of soft tissue and fluid-filled viscera, and the muscular and connective tissue structures of pelvic floor and urethra are well visualized. MRI can identify pelvic masses, ureteral obstruction, urethral diverticulum, pelvic organ prolapse, levator ani defects, anal sphincter defects, and other pelvic floor abnormalities. MRI is the imaging technique of choice for a suspected urethral diverticulum.

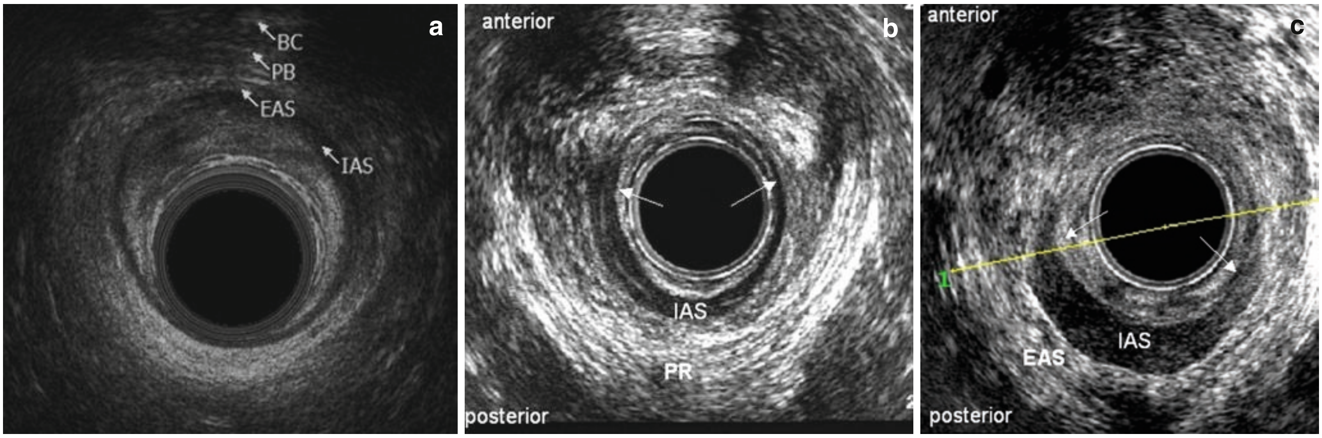

Fig. 5.6 (**a**) Normal endoanal ultrasound with intact external and internal anal sphincters. The internal anal sphincter appears as a hypoechoic circular band. The external anal sphincter is seen as a thicker echogenic band just exterior to the hypoechoic internal sphincter muscle. (**b**) Endoanal ultrasound with internal sphincter defect between 11 and 2 h (see *arrows* for sphincter defect) (**c**) Endoanal ultrasound with external sphincter defect between 8 and 3 h (with a lesion angle of 178°) and internal sphincter defect between 8 and 4 h (see *arrows* for sphincter defect). (Images courtesy of Sthela M. Murad-Regadas, MD)

Dynamic and standing MRI is a new technique for imaging of the pelvic floor, and there is still limited knowledge and data available on its clinical utility. The addition of dynamic (with relaxing and straining views) and standing MRI to observe pelvic floor descent in motion has improved our visualization of the anatomic detail of pelvic floor and anal sphincter musculature. Disadvantages of MRI are that it is costly, time-consuming, not widely available, and its results have not yet been validated or standardized. In addition for evaluation of the anorectal region, MRI requires specialized endoanal coils for optimal imaging.

MRI has been studied for the radiographic evaluation of stress incontinence. Urethral hypermobility and bladder neck descent are anatomic characteristics of stress incontinence that is seen on MRI. In addition, women with stress urinary incontinence have attenuated urethropelvic ligaments and an increased distance between the pubic symphysis and bladder neck on MRI.[32] The ability to visualize the position of the bladder neck and degree of cystocele at maximum Valsalva effort on dynamic MRI is equivalent to information obtained from lateral cystourethrogram. Currently, the indications and advantages of using MRI in the routine work-up and management of pelvic floor disorders have not yet been established.

Summary

A thorough understanding of a patient's incontinence and pelvic floor problems typically requires the appropriate use of complimentary studies including validated quality of life questionnaires, pelvic floor strength and tissue quality assessment, urodynamic investigations, cystourethroscopy, and neurophysiologic testing. A comprehensive understanding of a patient's genitourinary anatomy and function can be obtained through several imaging modalities that provide excellent anatomic and structural detail of the pelvic floor. Three-dimensional and dynamic ultrasonography and dynamic and standing MRI are new technologies that offer excellent visualization of pelvic structures and are currently being developed within research protocols to enhance our understanding of complex pelvic floor disorders.

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Part II

The Grafts

The Principles of Mesh Surgery

6

Peter von Theobald

Etiopathogenesis of Pelvic Floor Defect

The causal elements of a prolapse are multifactorial. Pelvic floor traumatisms as provoked by pregnancy and vaginal delivery are certainly very important. They are responsible for tissue elongation, nerve and vessel damage, elastic tissue breaks. Postmenopausal atrophy of the pelvic floor tissues is another well-demonstrated factor, frequently destabilizing a pre-existing injury. Obesity and chronic bronchial obstructive disease increase the risk of prolapse. But it seems to us that the main risk factor for pelvic floor relaxation is the quality of the connective tissue in the pelvis and the perineum. Many series are now available, assessing samples of uterosacral ligaments, vaginal tissue from the apex, from the anterior wall, from the Paraurethral position. Significant modifications are pointed out. For the apex, smooth muscle cells and collagen III as well as active matrix metalloproteinase 9 (MMP 9) concentrations are raised in POP.[1, 2] For the uterosacral ligaments, collagen density, collagen III and Tenascin concentrations are raised in POP with a decrease in Elastin.[3–5] One series[6] amazingly finds no significant change in POP in uterosacral ligaments as in vaginal tissue. But the site of vaginal tissue sampling is not precisely described in the paper. There are different variations in apex tissue and uterosacral ligaments compared to anterior vaginal wall or Paraurethral tissue, where collagen III, I and VI concentrations, Vitronectin expression, and extracellular matrix density are reduced in POP.[7–10] Another publication reveals reduced amount of smooth muscle cells in the round ligaments of patients with POP.[11]

All authors insist on the alterations of the extracellular matrix of the pelvic floor connective tissue associated with decrease of smooth muscle cells. The tissue of the fascias and the ligaments is less elastic and more breakable. The real question is: are these changes aetiology or consequence of the POP? Three publications tend to underline the primary weakness of the collagen in POP. A recent study[12] has shown positive correlation between low bone densitometry and pelvic organ prolapse (POP). As we know that osteoporosis is first a disease of the collagen matrix of the bone, especially of its turnover, we can imagine a similar mechanism for POP. Furthermore, a recent review upon SERM[13] shows very diverse effects of SERM on POP: Some, like Raloxifen, are protective, while some others have been retrieved from trials because of their POP inducing effects. Knowing that SERM are modifying the expression of several genes involved in collagen turnover and extracellular matrix integrity, we can argue that a dysfunction of these genes leads to connective tissue breakdown and POP. Another paper has established a strong association (OR3.12, $p<0.05$) between two connective tissue disorders: striae and POP.[14]

It appears that primary, genetically set bad connective tissue or premature ageing of this tissue may be the "primum movens" of POP. But we all know that small prolapses are likely to regress in time. A recent study has confirmed these data[15]: regression rate for grade 1 prolapses after 2–8 years is 23.5% for cystocoele, 22% for rectocele, and 48% for uterine prolapse. Progression rates are only 9.5%, 13.5%, and 1.9%, respectively. This means that many women undergo vaginal distension and distortion at the time of the delivery, but most of them are able to repair properly their tissues. Furthermore, undergoing the same mechanical stress, different patients may have various degrees of pelvic floor tissue injuries. The degree is correlated to the elasticity and the resistance of their connective tissue. Only a longitudinal prospective study taking samples in a high risk of POP population and comparing the results to the anatomical outcome after 5 or 10 years could determine which collagen profiles are predictive of further POP. Such a study would be difficult to organize.

To summarize this section, we can write that POP is due to distension and / or disruption of weak, fibrous, and inelastic connective tissue. A correct repair should focus on the anatomic correction of the disruptions or distensions and on the improvement of the supportive tissue.

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Principles of the So-called "Traditional Repairs"

Modern anatomists like DeLancey[16] and Petros[17] have perfectly identified the possible defects. Rupture or distension may be medial like a hernia through the fascia, sometimes focal, sometimes extensive. The injury may be lateral, detaching the vaginal fascia from the pelvic sidewall. It may also be an upper defect, if the fascia is detached from the pericervical ring. The consequence of this advanced anatomical knowledge was the birth of the "site-specific surgery of POP." The aim was to avoid a global repair, already known as painful and causing vagina narrowing. The site-specific procedure intended to reattach the disrupted fascia or ligament in its correct position, once the defect was perfectly diagnosed. The problem is that in most patients, defects are multiple. Thus, a site-specific repair frequently becomes more or less global. The more you have to pull on a weak, thin, and distorted fascia, using tension to repair it, the more it will get thinner, weaker, and likely to break in another place. Tension-full suture is always fragile and painful. Nothing was done to improve the quality of the tissues.

One of the principles of the classical techniques is to treat the colpoceles by excising the redundant vaginal wall tissue. Colpectomy was one of the main components of the repair procedures, aiming to narrow the vagina to its "normal size"! Probably, because the vagina is a virtual cavity, the colpectomies were frequently very extensive. The colporraphies, myorraphies, and perineorraphies were very tight, in order to let nothing come out of the vulva. Frequently also, these tight repairs were causing pain and dyspareunia.

Another principle of classic repairs is the hysterectomy. The fundament of gynecological surgery is the performance of hysterectomies, as many as possible. A removed uterus is not likely to prolapse anymore. Since the nineteenth century, hysterectomy was the treatment or part of the treatment of POP. It is very difficult to go against tradition, even when modern knowledge has shown that the POP is not a disease of the uterus.

Finally, let us ask the main question: what is the classic or traditional POP repair technique? "Mine, of course" you will say, and you are right. "Mine too," I will say. And your colleague's too. And the technique of the doctors you've been trained with, too. Obviously, everybody knows the traditional technique, all over the world, and sometimes they compare it to the new techniques. But there is a slight problem: none of these traditional techniques are the same. Colpotomies are different: horizontal, vertical, T-shaped, Y-shaped, diamond shaped.… Fascia dissections are very various: complete dissection, dissection from the vaginal wall, dissection from the bladder or rectum, no dissection.… Repairs are widely various: simple suture, overlapping suture, purse suture, multiple transversal sutures.… Apical suspensions are numerous: no suspension (very frequent, replaced by a very tight and high myorraphy), uni or bi-lateral sacrospinous ligament suspensions, vaginal high MacCall suspension, Ilio coccygeous muscle suspension, Other uterosacral ligament suspension.… Plus a thousand additive procedures, aiming to reinforce the repair, using detached uterosacral ligaments (Shirodkar, Manchester, …), pubococcygeous muscle (Lahodney and variations, myorraphies of several types), fascia and uterus (Fothergill and variations), round ligaments and vaginal wall (bridge repair, overlapping sutures …). The indications of all these techniques are empiric, more related to the surgeon's personal preferences and fashions than to any scientific knowledge. Even the impact of hysterectomy on POP has not been studied until recently.[18]

To summarize this section, we can say that the traditional repair is not standardized and that it is aiming through very various means to repair defects by stretching and reattaching autologous tissue, to narrow the vagina and vulva to prevent a descent of any kind and to remove rather systematically the uterus for the surgeon's pride.

Principles of Mesh Surgery

In the 1950s, a Parisian team led by Scali and Hughier, being aware of the high recurrence rate after vaginal repair of POP and abdominal repair using autologous tissue, published the first series of mesh repair by abdominal approach. Their aim was not only to suspend the vault or the uterus to the prevertebral ligament at the level of the promontory, as unfortunately later described by many (American) authors, but they were also intending to reinforce both anterior and posterior fascias, inserting a "hammock" in the middle of the pelvis from the pubis to the vault or the uterus and then to the levator ani muscles and the promontory.[19] They have set the first stone to the new POP surgery: *weak tissue reinforcement*. Correcting the anatomy is not enough; to maintain the result you have to improve the quality of the supporting tissue.

During decades, after surgical implantation, the mechanical properties of the mesh were supposed to provide the strength necessary to support the pelvic organs. This belief still persists in many minds; a strong mesh, anchored by strong nonabsorbable sutures is thought to be the ideal repair. Against this, Michel Cosson's publications[20] on the resistance of the various pelvic fascias and ligaments have demonstrated the poor quality of our traditional fixation structures. Tendinous arches, uterosacral ligaments, and even sacrospinous ligaments are weaker than the softest mesh or band we are using. Thus we have to conclude that the hold of the POP repair is not depending on the resistance of the mesh nor on the nature of the sutures attaching it to the fragile structures of the pelvis. We are not aiming to suspend the genitals to a strong hook with a tough rope, because there is no strong hook and a tough rope would involve local complications as erosion, pain, and fibrosis. Three main principles are leading to a successful repair with meshes: *the bio surgery of the collagen* (or directed healing process), the *correction of the anatomical axes* of the vagina, and *the tissue-sparing approach*.

Bio surgery of the collagen: this concept has been invented by Hubert Manhès in the early 1990s, when he intended to correct cystocoele by laparoscopy with a mesh in the Retzius space held only by fibrin glue and a pessary. In fact, when you insert a mesh in any tissue, it will involve a foreign body reaction. Fibrin due to the dissection will quickly surround the mesh, giving a soft primary hold. Granulocytes and macrophages will colonize the mesh within 48 h in the process of the inflammatory response. After a week, fibroblasts will appear on the mesh, starting to produce the extracellular matrix of a new connective tissue around the mesh. Thus, the goal of the mesh is mainly to "stake" the building of this fresh new connective tissue in the correct dissection plane, in the right direction. This tissue, being continuous with the pelvic fascias and ligaments around it, will be more resistant to tearing and traction than any suture could be by itself. The tissue in growth of the mesh is the key of resistance, not the mesh itself.

Since connective tissue is a live tissue, it needs to be continuously remodelled to stay resistant and elastic. The proteic matrix needs to be renewed especially in these POP patients, whose collagen is of poor quality (if it was strong, they would not have developed a POP). If an absorbable mesh is inserted, it will induce an inflammatory response followed by macrophages and fibroblasts, and a new fascia or ligament will be produced on this site. But when the mesh is finally absorbed, the new collagen will age and undergo the natural evolution of the autologous one, which is poor. If a nonabsorbable mesh is used, the foreign body reaction will persist and the collagen will be renewed on a regular basis around the mesh and the quality of the new ligament or fascia will stay constant.

Another very important feature of the prostheses has to be underlined here: the pore size. Implantation of a polypropylene thread provokes a foreign body reaction with fibrosis around it. The average extent of this fibrosis is X micron. If two threads are too close (distance < X × 2), the fibrosis will be continuous, involving rigidity and retraction. On the contrary, if the distance between the threads exceeds X × 2, space for normal soft tissue is left in the pores (Figs. 6.1 and 6.2). Thus, elasticity is preserved and mesh shrinkage decreased. The complication rate also seems to be reduced with larger pores. As the result of the repair is not due to the resistance of the mesh itself, but to the neofibrogenesis involved, large

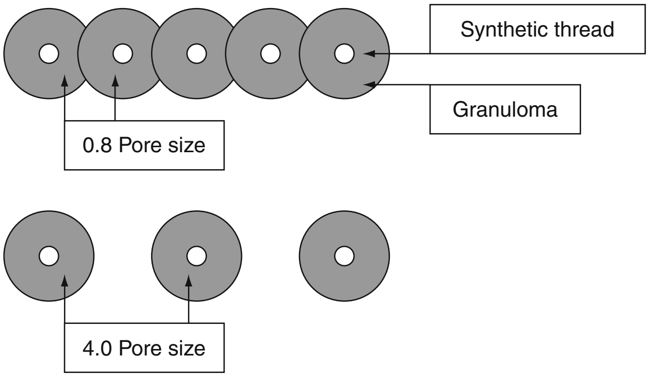

Fig. 6.1 Difference between small pores and large pores in polypropylene meshes: peri-filamentous fibrosis is bridging in small porous meshes and not in large porous ones. Bridging prevents tissue ingrowth and induces fibrotic retraction of the filaments and surrounding tissue[21]

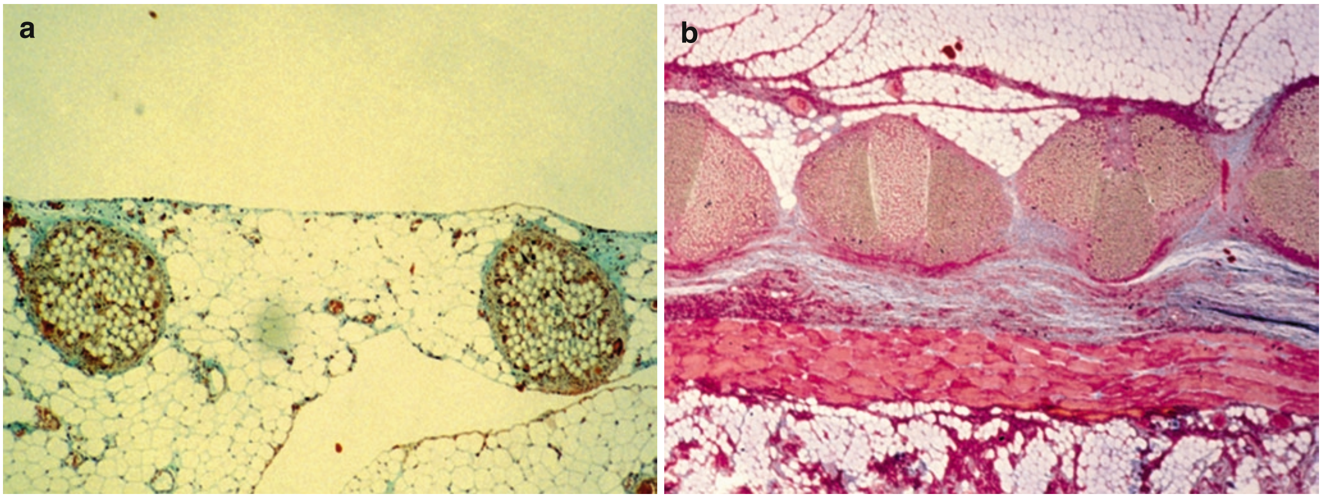

Fig. 6.2 (**a**) Large pores with normal tissue ingrowth; (**b**) small pores with bridging

pore sizes can be used. The lightest meshes available today have pore sizes between 0.5 and 1.00 mm. The limit is not known to date; it will probably depend on surgical conditions. A very light mesh will be difficult to handle, to insert, and to fasten to the pelvic sidewall. It will certainly require some absorbable coating to make it easier. But this coating should not enhance the early inflammatory response as the actual synthetic absorbable materials, like Polyglactin, do. Maybe, this could be a very interesting indication for the use of biomaterials.

The Correction of the Anatomical Axes of the Vagina

The vagina is a very sophisticated anatomical structure (Figs. 6.3 and 6.4) and its perfect reconstruction is a real challenge. But the results of pelvic floor surgery depend on this anatomical correction. The vagina starts at the vulva as a vertical slot, crosses the elevator plate aiming toward L5–S1 and then the posterior vaginal wall lies on the elevator plate, aiming toward S3. The vagina ends as a horizontal slot. Thus, increasing intraabdominal pressure at effort will apply the horizontally flat upper third of the vagina on the elevator plate (if it is in the right place, maintained by the utero sacral and cardinal ligaments). Meanwhile, the puborectalis will increase the angulation between upper and lower third of the vagina, compressing laterally the vagina and closing the vertical slot portion.

This functional anatomy is related to balanced forces as described by Peter Petros in his Integral Theory.[17] Pelvic floor repair should aim to restore these forces without any over tensioning. Overcorrection of one segment will always lead to pain, dysfunction, or breaking involved by increased tension on another segment.

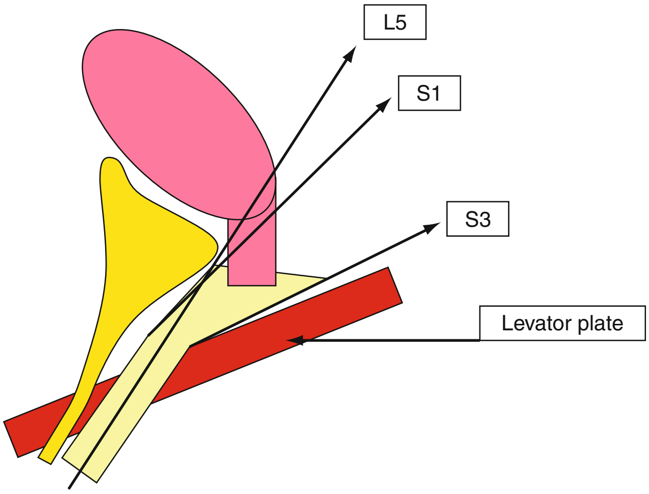

Fig. 6.3 Sagital axes of the vagina

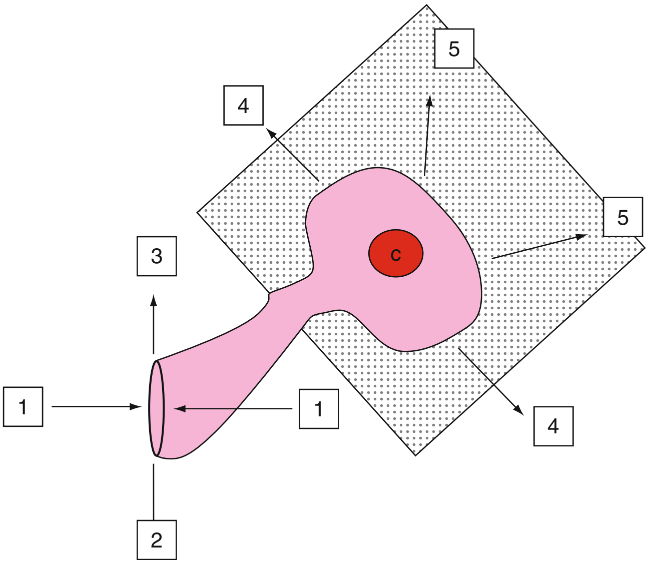

Fig. 6.4 Three-dimensional view of the vagina. (*1*) Bulbocavernosus ans puborectalis muscles; (*2*) perineal body; (*3*) pubourethral ligament; (*4*) cardinal ligaments, paracervix and parameters; (*5*) uterosacral ligaments

The Tissue-Sparing Approach

This approach concerns two aspects of the traditional surgery; the belief in the need of a systematic hysterectomy and colpectomy. Prolapse is not an illness of the uterus and hysterectomy does not cure the prolapse. The apical defect has to be treated specifically or the POP will recur. Hysterectomy increases morbidity[18] without evidence of increasing success rate.

Colpectomy aims to trim the excessive vaginal epithelium in order to "tailor" a "normal looking" vagina. But vaginal epithelium is a live tissue; it is able to recover after incision and distension. For instance, after distension by vaginal delivery, it retracts back to its normal size after a couple of hours. After abdominal or laparoscopic sacrocolpopexy (almost never associated with a colpectomy), it retracts to a normal shape within a few days. Of course, at the end of the surgery, a trimmed vaginal wall looks better because it is not redundant. But, as soon as the patient rises from bed or when the bladder or the rectum fills, it is under tension and its thickness is reduced as is its vascularization and innervation. This is crucial if you use meshes to reinforce the fascia; a local necrosis of the overlying epithelium will expose the prosthesis and result in erosion. It may also be a problem in conventional surgery and explain some of the frequent recurrences mainly in cystoceles; sometimes, the vaginal mucosa is so thin that you almost can see the bladder through it. The main difference between traditional vaginal POP surgery and mesh surgery is that the first is treating anterior and posterior colpoceles and the latter is treating cystoceles and rectoceles.

Who should benefit from mesh surgery? Only recurrence cases? In this strategy, only a few procedures would be performed and on patients in whom the dissection is the most difficult due to fibrosis. This involves poor results because performing few cases on specially sophisticated patients means that the procedure will not be standardized and makes the learning curve much more difficult as with easy primary patients. It is like learning laparoscopy on rectovaginal endometriosis patients instead of progressive education starting from extrauterine pregnancies. Only patients with risk factors for recurrence ? This strategy looks better, but the risk factors still have to be clearly established. In literature, recurrence occurs in more than 30% of patients, requiring a second operation. In all prolapse patients? The aim here is to have a perfectly standardized technique, an easy learning curve and to offer every patient a painless, minimally invasive, long-term effective operation. This is the protocol applied in the University Hospital of Caen since June 2001 including every case of the 250 POP operations every year. Who should not benefit from mesh surgery ? Infected patients or those at high risk of infection (instable diabetes, immunodeficiency, …): certainly. Young patients? Some surgeons may have concerns on inserting meshes in patients under 50, wondering on the future of the mesh after 20 or 30 years. They will apply a traditional, vaginal narrowing technique with a high risk of recurrence in this very active patient. This seems to be illogical; young active women need a long-term effective technique preserving the totality of their vaginal tissue to avoid dyspareunia. Only mesh surgery can provide this. Would you insert breast prostheses or hip prostheses only in women above 70 and try an old-fashioned traditional repair with autologous tissue before?

Conclusion

The aim of using mesh in prolapse repair surgery is to use the foreign body reaction to reconstruct good fascias and ligaments in an orthotopic position and to maintain them by promoting the renewal of their collagen. According to the recent advances in anatomical knowledge, mesh surgery allows tension-free physiological repair, resulting in almost pain-free surgery and excellent long-term results. Mesh surgery of POP is organ and epithelium-sparing to reduce the surgical traumatism, to improve function, and to promote a quicker return to normal activity.

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7

Properties of Synthetic Implants Used in the Repair of Genital Prolapses and Urinary Incontinence in Women

Michel Cosson, Philippe Debodinance, Jean-Philippe Lucot,
and Chrystele Rubod

Introduction

Alterations can often be seen in the structure of the biological tissues of patients suffering from disturbances of pelvic function, in particular, with respect to the properties of their collagen.[1–4] There is, therefore, a great temptation to use reinforcing materials during surgical treatment of genital prolapse and strain urinary incontinence. Up to now, most observations on synthetic prostheses have come from the field of reconstructive surgery of the abdominal wall and the repair of groin hernias.[1,5–8] Implants have been used in gynecology for the last 20 years or so for transabdominal prolapse repair and in urinary incontinence through suburethral slings.[9–12] More recently, the unresolved problem of the treatment of cyctocele has led surgeons to try to use these materials by vaginal route, even though it is reputed to carry a risk of infection.[13–15] A large number of products of biological and synthetic origin have been proposed for this application. We shall be voluntarily restricting ourselves here to examining synthetic reinforcing materials and considering their composition and mechanical and other properties when such data are available. We also address the outcome after implantation, as regards tolerance. The properties and composition of the implants are well known and the recent history of severe complications relating to polyester, silicone, heat-welded polypropylene, and multifilament implants highlights the risk that a negligent attitude in this field can produce. An international standard applicable to the marketing of any new materials would be most welcome, along with consensus as to the prior animal and clinical experimentation required.

Description and Properties of Synthetic Prostheses

Absorbable Synthetic Prostheses

There is a very real temptation to use absorbable prostheses to reinforce surgery of the pelvic floor. These implants cannot undergo secondary rejection and their presence favors postoperative fibroblast activity. This enthusiasm has, however, been tempered by a number of studies.[7] The most commonly used absorbable prostheses are Dexon® (polyglycolic acid) and Vicryl® (polyglactic acid).

The absorption of absorbable prostheses should be slow. Vicryl® begins to be absorbed at the beginning of week 3 and by the 30th day, only a few elements remain, with no mechanical value. The mesh is absorbed through the action of macrophages and it is replaced by healthy scar tissue. The absorption products are recycled into new collagen fibers.[16] Dexon® is absorbed in 90 days. The scar tissue is never as strong as reinforced tissue and alone is insufficient to provide the strength of the structure. Absorbable implants are ephemeral by nature and are not therefore threatened by infection. They are not harmful to the viscera. A study by Lamb in 1983[17] demonstrated that the fibrous reaction was insufficient before absorption of the implant. Tyrell[18] created and repaired a ventral lesion in rabbits and secondary hernia was noted in 40 of those treated with an absorbable implant. The results were confirmed by Rauth,[19] using Vicryl implants in rabbits. A study carried out by Brenner in 1994[20] also confirmed the findings and further clinical evidence has since been provided by several randomized prospective studies. The results underpin, if there were still any need to do so, the poor results obtained in prolapse repair by simple excision and tightening of the excess vaginal tissue of the vaginal tissue without suspension.

To conclude this chapter, it appears that the literature is unfavorable to the implantation of absorbable prostheses for prolapse repair by the vaginal route, based on both animal studies and clinical results.

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Nonabsorbable Synthetic Prostheses

Here, we offer a brief overview of the development and history of prosthetic materials, their properties, and experience of their use in gynecological surgery.

History of Prosthetic Materials

1. Metal Meshes
   (a) *Silver mesh*: Phels was the first to come up with the idea of a silver mesh, in 1894. The concept was subsequently developed by Wizel, a German surgeon, in 1900 and exported to the USA from 1903.[5] Silver mesh corroded rapidly on contact with tissue fluids.[6]
   (b) *Tantalum*: Tantalum is as inert as glass and its use began in 1940. The numerous irregularities, however, caused fragmentation and intestinal fistulae and metal fragments were found in the abdominal cavity and in the cutaneous covering.[6]
   (c) *Stainless steel*: The first use of stainless steel as a prosthesis was by Babcock[7] in 1952. Haas and Ritter developed a chain of stainless steel rings 10–11 mm wide and 4 mm in diameter, based on a ring chain net made of silver wire, designed by Goepp in 1928.[8] The infection rate was 0.1%, few complications were reported, and the results seem to have been favorable.
2. Nonmetallic Synthetic Prostheses
   (a) *Fortisan cellulose fabric*: Fortisan fabric was a cheaper, more malleable alternative that offered good infiltration by host tissue, but was rapidly shown to be a source of infection and fistulae.
   (b) *Polyvinyl sponge*: (Ivalon®, 1949) This is a polymer of polyvinyl alcohol in its formaldehyde form, in which air is blown through a liquid plastic to form a solid appearing like a cross-section of a slice of bread. It was, however, poorly tolerated during infection, and had a tendency to dissolve and fragment with time.
   (c) *Nylon*: Nylon was developed in 1938 by Dupont de Nemours, and became hugely popular after the Second World War, when it had been used for making powder bags for the US Navy and parachute cloth. Its first surgical use was as Crinoplaque®,[21] which was woven from a tubular monofilament. This was followed by interlock knitted nylon, giving a fabric that would not fray and which could be cut using scissors. Although this was an improvement, the interlock form exhibited progressive alteration in situ and was superseded by other polymers.
   (d) *Silastic*: This polymer was combined with Dacron or nylon mesh and sandwiched between two layers of silicone. It was primarily used in pediatric surgery for the temporary closure of congenital defects.
   (e) *Téflon*®: Polytetrafluoroethylene (PTFE). This mesh is not incorporated into body tissues. In 1964, Gibson reported a 50% rate of parietal complications.[22] Teflon's poor tolerance to infection was also reported by several other authors.
   (f) *Carbon fiber*: Carbon fiber was developed from 1980, but has been little used. A composite made by immersing carbon fibers into a solution of dilute chloroform-treated polyglactic acid provided better biocompatibility. Its durability is identical to that of polypropylene.
   (g) *Polyester mesh*: *Dacron*. Dacron actually stands for a variety of products composed of a saturated polyester, polyethylene terephthalate (PETP).[23] It originally appeared in 1956 and is also known as Mersilene (Mersuture®) when knitted and Ligatene® when woven. Dacron mesh has been the most popular and most utilized nonmetallic mesh of the last 4 decades, but its use is currently on the decline. A silicone-velour prosthesis, the Rhodergon® patch[23] started being used in the 1970,s but was rapidly withdrawn as its impermeability made it unsuitable for the parietal environment. It should not be confused with the Rhodergon 8000® mesh, which is made of polyester supplemented with a felt, giving it the appearance of an inextensible patch, but which is permeable.
   (h) *Polypropylene (PP):* In 1958, Usher[24] introduced Marlex®, a fiber formed by weaving a single monofilament of polypropylene. It was so popular that from 1962, 20% of hernias in the USA were being treated with Marlex mesh. However, the use of Marlex was associated with complications in cases of abdominal and especially exudative sepsis, and others such as exposure of the implant (44%) and digestive fistulae (23%), and it was withdrawn. Prolene®, an equivalent product made with two filaments, and the multifilament Surgipro®, give good overall results with minimal complications.
   (i) *Expanded polytetrafluoroethylene (ePTFE):* ePTFE, otherwise known as Gore-Tex®, was discovered in Japan in 1963 and is an expanded form of Teflon, which makes it microporous and, unlike Teflon, allows it to be incorporated into the tissue. Gore-Tex® is stronger than Marlex®, Prolene®, and Mersuture®, and is incorporated more rapidly and with minimal inflammatory reactions and few adhesions. It is currently widely used in parietal surgery, notably in surgery of a provisional nature, such as in laparoschisis.

Classification of Biomaterials

The prevention of complications requires in-depth knowledge and understanding of the physical properties of the implants, of which porosity and pore size are of major importance.[25] The classification system devised by Amid[25] is currently the standard (see Box 7.1). This historical classification has, however, lost some of its relevance as the vast majority of manufacturers market only polypropylene implants, which means that a classification made up purely of this type of prosthesis could be introduced. This classification, which we shall be discussing further on, would be based on the Amid classification, taking the nature of the materials' composing fibers (mono- or multifilament), their mean porosity, and their density.

Box 7.1 Amid Implant Classification

Type I: Completely macroporous mesh (Atrium®, Marlex®, Prolene® and Trelex®). The pore size exceeds 75 µm, the size required for infiltration by macrophages, fibroblasts, blood vessels in angiogenesis, and collagen fibers.

Type II: Totally microporous mesh (Gore-Tex®, surgical membranes). The pore size is smaller than 10 µm in at least one dimension.

Type III: Macroporous patch, with multifilaments or a microporous component: PTFE (Teflon®), woven Dacron (Mercilene), woven Polypropylene (Surgipro®), perforated PTFE (Mycro Mesh®).

Type IV: Biomaterials with submicronic pores (Silastic, Cellgard®, dura mater substitute). These materials are often associated with those of type I to prevent adhesion in intraperitoneal implantation.

Source: Data from Amid K.[25]

Implant Structures

The main, currently used synthetic implants are made of polyester, PTFE, polypropylene, polyethylene, and nylon. Their mechanical properties depend on the structure of the fabric and the thread. *Woven* implants can be plain, twill, or satin weave (Fig. 7.1). Their advantages are stability and good memory. Their disadvantages are fraying and poor conformity. *Knitted* implants can be warp-knit, interlock, and circular-knit (Fig. 7.2). These fabrics are flexible on manipulation, versatile, and have high conformity. The unwoven materials are absorbed well, but have no conformity, are poorly visible, and demand a higher level of treatment. *Composite fabrics* have two surfaces, one of which prevents adhesion and synergistically enhances the properties of each element, but they are rigid and poorly visible. They can have holes, be kidney-shaped, umbrella-

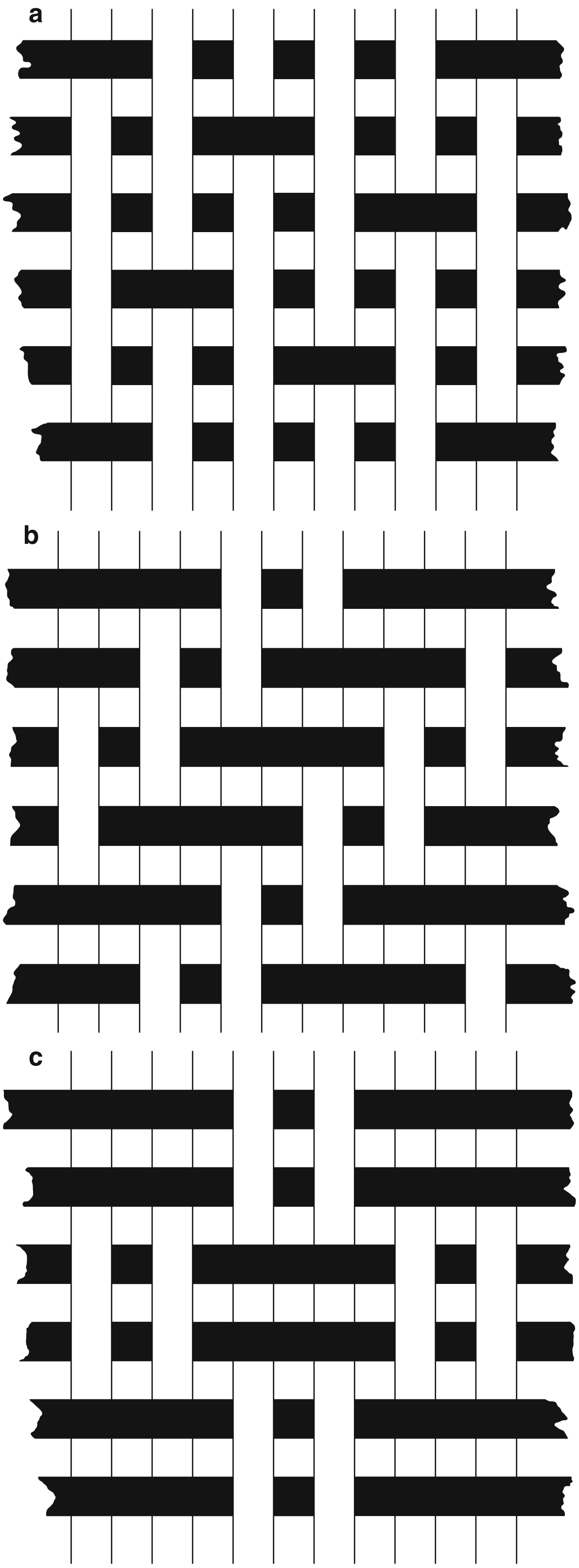

Fig. 7.1 Woven fabric (**a**) Smooth, (**b**) Crossed, (**c**) Satiny

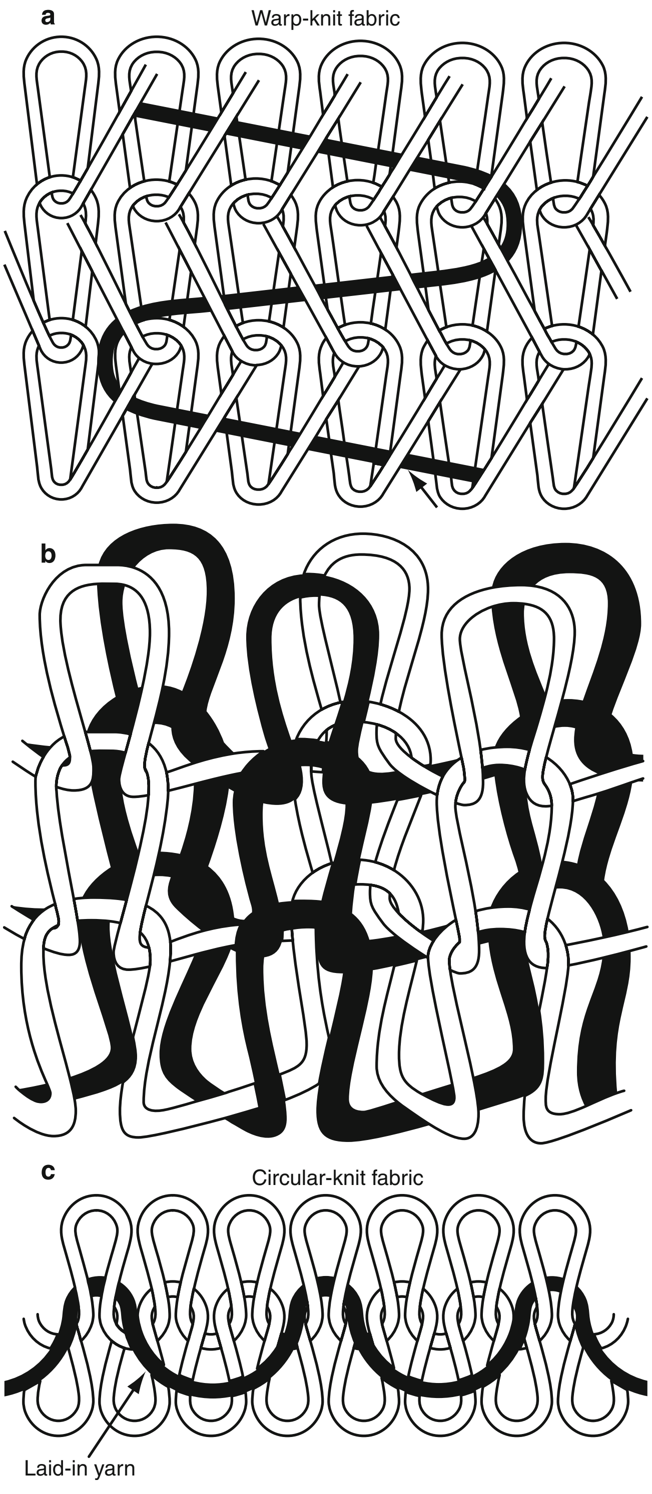

Fig. 7.2 Knitted fabric (**a**) Chaîne tricotée (**b**) Procédé interlock (**c**) Circulaire

shaped, or in the form of a plug. The fiber structure can comprise a monofilament or multifilament, can be twisted, coated (by one or two layers), braided, or double braided (Fig. 7.3).

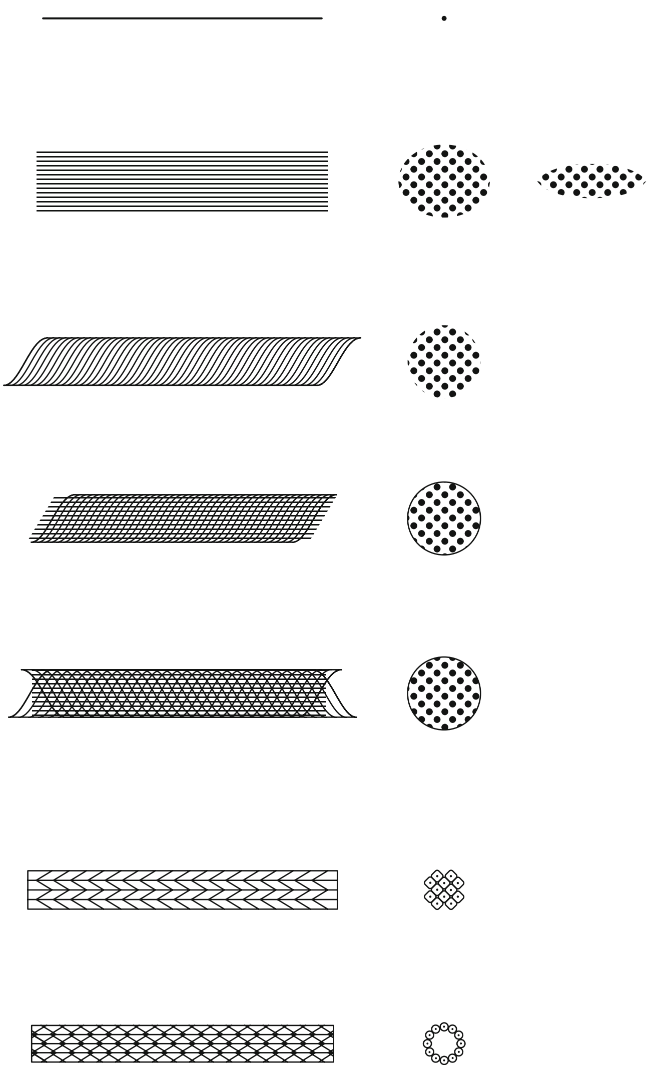

Fig. 7.3 Fiber structure. From *top* to *bottom* (Monofilament, multifilament, twisted, coated, double coated, double layer, braided)

Mechanical Properties and Tolerance of Synthetic Implants

Mechanical Properties

These properties are directly dependent on the type of thread and the knitting method used in the implant. There are no recommendations concerning the resistance or elasticity of implants used in hernia or prolapse surgery. The information provided by manufacturers is incomplete and more comprehensive data need to be provided.

With the lack of manufacturer data, there is no established scientific link between the implants' mechanical properties and tolerance and their clinical outcomes. The mechanical properties of the implants, arbitrarily determined for utilization in hernia and abdominal eventration repair, are not designed to be used in genital prolapse repair. These polypropylene implants are too rigid and dense for the pelvic tissues they are used to reinforce and the minimum porosity and density need to be determined more accurately and more scientifically.

Biological Properties: Development After Implantation

The reaction of the soft tissues to implanted biomaterials is variable.

Generally speaking, nonabsorbable synthetic implants provide ongoing and renewed local stimulation of cicatrisation. Williams[26] identified four types of response:

- A *minimal* response, with a thin layer of fibrosis around the implant.
- A *chemical* response, with a severe and chronic inflammatory reaction around the implant.
- A *physical* response, with an inflammatory reaction to certain materials and the presence of giant cells.
- *Necrotic tissue*: a layer of necrotic debris is produced, resulting from in situ exothermic polymerization.

The first type of response consists essentially of normal scar formation at a wound, in which a thin layer of fibrosis isolates the implant from surrounding tissue. PTFE, polyethylene, and silicone patches produce this reaction. PP, PGA, PETP, and PTFE have good biocompatibility properties. For example, PTFE has a low critical surface tension, which prevents the attachment and propagation of cell growth, thereby limiting cell penetration into the pores of the knitted structure. Conversely, PETP and PGA, which have numerous ester groups, provoke an acute thrombotic response that encourages fibroblastic cells to attach and proliferate along the filamentous surfaces. PP results in a less acute cellular response than PETP and is therefore recommended for reinforcement of the inguinal canal, or for covering sandwiched peritoneum in the repair of incisional hernias. PP mesh is not recommended for integrated implants, due to the elevated risk of visceral adhesions in the abdominal cavity.

Kaupp[27] described a histological reaction in four stages:

- Stage 1: In the first week, the symptomatology comprised an intense inflammatory infiltrate around the implant, capillary proliferation, granular tissue, and the presence of giant cells containing birefringent material.
- Stage 2: After 2 weeks, the granular tissue remained, spumous histiocytes had appeared, with more or fewer giant cells.
- Stage 3: After 4 weeks, the acute inflammation had disappeared, capillaries were reduced, and the number of spumous histiocytes and giant cells had increased.
- Stage 4: Some giant cells were present on the external surface of the implant and a dense, fibrous tissue was present.

The reduced inflammatory reaction provoked by the nonabsorbable parietal implant was a sign of it being well tolerated by the host, and the intense fibroblastic reaction showed that it was becoming integrated into the surrounding tissue. Under normal conditions, the inflammatory reaction, initially an exudate, then cellular, starts on day 3 and the fibroblastic reaction which supersedes it and colonizes the implant begins around day 10.[28] The relationship between the numbers of inflammatory cells and fibroblastic cells allows a simultaneous evaluation of the mechanical value and the biological tolerance of the implant by the host.[29] A Dacron mesh implant is completely colonized and integrated into the abdominal wall after 4–6 weeks.[29] It is important to note that the critical period for the stability of a parietal implant, just as for the strength of a parietal stitch, is between days 7 and 10. The strength of a prosthesis increases with time: in an animal study, it doubled between weeks 3 and 12 for PP or PTFE.[30] The major risk is an early infection. Butha[31] has shown noninfected collections around Dacron velour implants, suggestive of an allergic reaction. This theory cannot, however, explain why the rejection is not always bilateral. Kaupp[27] claimed that periprosthetic reactions are not due to infection, but showed macroscopic features compatible with an immunological reaction. De Clerk[32] accepted the theory of delayed hypersensitivity and foreign body reaction in the host suggested by Kaupp,[27] since he observed an improvement under cyclophosphamide. This immunological mechanism is difficult to prove, because no test exists and it is not possible to discriminate histologically between an immunological reaction and a simple reaction to a foreign body. Studies by Katz[33] have addressed the concept of the hydrophobicity of biomaterials, and shown its influence on bacterial adhesion. These properties can also influence the dynamics of absorption and resorption of the organisms on the surface of synthetic materials. Bacterial adhesion is initially a reversible process, and becomes irreversible when the bacteria create an extracellular adherence. On removal of 18 implants, Klinge[34] noted the presence of 32% of inflammatory cells in PP meshes, 12% for those made of ePTFE, 8% for polyester, and 7% for reinforced PP. He also noted the presence of macrophages at the interface between the tissue and the PP (45%), polyester (45%), ePTFE (25%), and reinforced PP (22%).

Clinical Use and Complications of Prosthetic Implants

Use of Meshes in Gynecology

Most articles refer to the use of meshes in gynecological surgery over the last 30 years. The implants have mainly been used for transabdominal prolapse repair (sacrocolpopexy) and suburethral slings. It is not possible here to detail all the articles published on the use of meshes in the repair of genital prolapse and urinary incontinence in women.

Transabdominal prolapse surgery, in particular sacrocolpopexy, has resulted in little or no intolerance, with only a few cases mentioned in the literature.[35] A number of articles discuss sling placement using a mixed vaginal and abdominal route, with vaginal and suprapubic scarring, often of very small size. Very few articles initially reported on an exclusively transvaginal approach, but the situation has changed now. Most of the cases involved interventions to provide support, as described by Mouchel.[36,37] In a review of the literature from 1950 to 1996, Iglésia[38] collated 21 retrospective, nonrandomized studies using slings and 15 sacrocolpopexies using synthetic material. The slings had been removed in 35% of cases and fistulae were noted in 10%. Sacrocolpopexy resulted in erosions in 9% of cases.

The most frequent symptoms[39] were pain, vaginal discharge, bleeding, induration of the abdominal scar, granuloma of a vaginal scar, abdominal or vaginal fistulae, failure of scar formation, and expulsion of the implant. Many implant rejections occur during the first year, but the period of follow-up is not often long. This may explain the low percentage of rejections reported in certain publications (0–39.8%) following interventions by the vaginal route or combined routes.

Norris[40] claims that the smaller the surface of the implant, the fewer the intolerance reactions. He noticed that the surface area of the synthetic material that he used (10.5 cm^2) led to six times less rejection than reported by Bent[41] using patches measuring 60 cm^2. This suggests a reaction proportional to the area of contact with the foreign body. Our experience has indicated the same reaction.[37] The area of contact between the foreign substance and the exposed tissue is an important determinant of the tissue reaction.[42] It could therefore be said that tolerance to the synthetic material is proportional to the exposed surface area and the distance that separates it from the vaginal scar.

TVT (Tension-free Vaginal Tape) should be discussed separately, even though it could be integrated into a sling intervention. A few remarks need to be made about the remarkable tolerance showed by this Prolene® strip.

Defective scar formation is reported in 0.5–2% of observations.[43,44] Nilsson[45] describes a case of sepsis of the vaginal wound. Usually, this is corrected by debriding the scar, minimal resection, and a new suture under antibiotic treatment. Tamussino[46] has described the Austrian registry of 7,000 TVTs and reported no cases of intolerance. This technique is minimally invasive and seems perfectly well tolerated, due to the small vaginal incision and protection of the strip by plastic-coated sheathes during its insertion.

The problem of transvaginal prolapse repair, notably for cystocele, remains to be discussed. While meshes have been introduced by the abdominal route since the 1970s, the use of the vaginal route has been much more recent and the first descriptions started appearing in publications only in the last decade. In prolapse repair, and especially for cystoceles, by the vaginal route, PP is the most frequently used material, and intolerance is marked by erosions and delayed scar formation, which were present in about 6% of cases (2–12%). Complete removal of the mesh was necessary only in rare cases. Partial resection and local treatment were sufficient and did not compromise the anatomical result.

Complications in Hernia Surgery

Infection

When the size of the pores or gaps in the mesh are less than 10 μm in each of their three dimensions, bacteria, which measure around 1 μm, cannot be eliminated by macrophages and neutrophils, as they are too big to infiltrate the pores. It has been shown that macrophages can actually find a way through the pores, but smaller pores do seem to correlate to a higher risk of infection. Meshes of types II and III are vulnerable to infection, since they may harbor bacteria and allow them to proliferate. Type I implants however, although they can be a refuge for bacteria, also admit macrophages and, more importantly, fibroblasts and angiogenesis, thus preventing infiltration and development of germs. Chronic infections occurring with materials of type I are mostly due to the use of multifilament stitches for attaching the mesh (10–50%). In the case of infection, removal of the implant is not necessary for type I materials, whereas, it should be completely removed if using type II and partially removed in the case of type III materials.[47,48]

Exudation

This is caused by an inflammatory reaction of the host to the implant and the space between the two. The faster fibrin binds to the implant, the quicker the space is filled, and the less the reaction appears. When the mesh is not in direct contact with subcutaneous fat, but in a retromuscular or infra-aponevrotic position, there is no exudation.

Intestinal Adhesions

Intestinal adhesions occur only with type I meshes in direct contact with the intestine.[49]

Erosions of the Hollow Viscera and Fistulae

Erosions of the hollow viscera and fistulae are also a complication of type I materials in contact with organs with or without a serous membrane and is not reduced by using absorbable materials.[49]

Retraction

Prosthetic plugs can retract to as much as 75% of their original size. This therefore predisposes to recurrence. Meshes retract during scar formation by about 20–30% of their surface.[50]

It can be clearly seen here that the complications described in transvaginal utilization of these implants had already been reported, but were largely ignored when the suburethral strip of type 2 or 3 implants were marketed.

Complications in Genital Prolapse and Urinary Incontinence Surgery

Complications of vaginal implants are certainly overestimated, sometimes demonized, often poorly described, and probably ill managed.

The complications are overestimated as it is much easier to publish a complication, especially a spectacular one, in a major urological or gynecological journal rather than a long prospective study of a few hundred patients; which, however, is the only way to estimate the actual frequency of the complications. The six randomized comparative studies that have been published, even while they do not show any more complications in the arm with implants than in the arm without, do not have sufficient discriminatory power. While awaiting results from large national registers, we noted all the serious complications reported in three studies: a Scottish study on 289 patients,[51] a Scandinavian study involving 148 patients,[52] and the results of the French TVM group containing 1,541 patients (unpublished data). Out of a total of 2,078 patients, there were 55 reports of severe complications, not including local implant resections for vaginal exposition (2.6%), breaking down as follows: hemorrhages>500 mL or significant hematomas (1.2%), deep infections (0.9%), visceral erosions (0.1%), and implant removal for complications (0.9%). Similarly, taking all the studies published on Prolift™, a total of 1,882 interventions, the percentage of each of the serious complications never exceeds 2.5%. The maximum reported rate of implant exposition is 12%, implant retractions 17%, and a de novo dyspareunia rate of 9%. We shall come back to the sexual consequence of the interventions later in this article. In our opinion, while all the surgeons (and of course their patients!) need to be aware of these complications, they do not appear to be overly discouraging.

Sometimes, patients are referred to surgeons who do not use the mesh technique themselves, but who treat patients with mesh complications (sometimes severe ones), because of their reputation as a traditional surgeon. Most of these complications could probably have been avoided by a proper initial surgical technique.

While it is true to say that the functional and especially the sexual consequences of the interventions were not analyzed in any detail in the first publications, this is no longer the case. The few randomized studies that do exist show that patients fitted with supportive vaginal implants do not show any more postoperative dyspareunia than patients operated on using a conventional technique.

They are often poorly described, as there is no clear objective and consensual classification. This is because it represents a new semiology that we analyzed over several years (the French TVM group has been studying outcomes and complications since 2000) before conceptualizing it. The outline is given below.

Three major groups of complications need to be drawn up systematically and in detail. We have separated complications caused by infections and which are often of a spectacular nature, from vaginal and other erosions which are the most frequent and retractions, both symptomatic and otherwise, which are the most difficult to treat and the most poorly described. Our simplified classification of the complications is given in Tables 7.1–7.3. (see also Box 7.2)

Table 7.1 Complications type 1: Infection of the implant

Grade	
1	Vaginal exposition with infection
2	Infection along with implant
3	Skin erosion near issue of the mesh
4	Local abcess
5	Distant abcess
6	Fistulate
7	Acute infection: Pelvic cellulitis

Table 7.2 Complications type 2: Exposition of the implant

Grade	Localization	Size
1	Vaginal exposition behind the incision	less than 0.5 cm^2: Few fibers visible
2	Vaginal exposition behind the incision	less than 1 cm^2
3	Vaginal exposition behind the incision	Over 1 cm^2
4	Vaginal exposition distant to the incision	Vaginal cul de sac
5	Erosion of an organ	Bladder, rectum, urethra, skin

Table 7.3 Type 3: Classification of the symptomatic contractions of the implant

Grade	Symptoms	Level	Area
1	Asymptomatic		Degree of retraction
2	Provoked pain only (during vaginal examination)		A: <1/3 B: >1/3, <2/3
3	Dyspareunia	Occasionally: + Usually: ++ Always: +++	C: >2/3
4	Pain during physical activities	Occasionally: + Usually: ++ Always: +++	
5	Spontaneous pain	Occasionally: + Usually: ++ Always: +++	

Box 7.2 Classification of Implants

The classification of implants devised by Hamid is therefore no longer relevant as the vast majority of supportive implants are type 1 knitted monofilaments of polypropylene. The most important task now is to gather data on the mechanical properties, both before and especially after implantation of supportive implants, on pore size, mass per cm², and thread diameter for subsequent correlation with the rate of complications or the anatomic outcome.

Type 1: Infections

Vaginal erosion, infection along the whole of the implant, abscess, cutaneous fistula. These are exceptional and always require the implant to be completely removed. They are generally caused by the implant material and are very rarely reported now that the use of knitted monofilament PP has become standard.

Type 2: Erosions

Type 2 complications. These are the most frequent and are sometimes termed expositions. They involve vaginal exposition of the implant, which can either be seen or palpated in the vagina or an adjacent organ. Vaginal scar erosions, or vaginal exposition, are the most frequent forms. A number of publications have indicated that prevention of these erosions is based upon keeping the uterus, whenever possible, limiting vaginal resections, and possibly performing small vaginal incisions. Patient weight and cigarette smoking are also thought to be contributory factors to the complications. Their physiopathology is poorly understood and occurrence varies widely from one hospital to another and indeed from one surgeon to another, with experience certainly being a key factor. Medical treatment is tried in the first few postoperative weeks and, if unsuccessful, minimal resection of the exposed implant is performed a few weeks later. These are not severe complications and treatment can be carried out under local anesthetic, but the symptoms can sometimes cause discomfort as they combine contact dyspareunia, metrorrhagia, or leucorrhoea. These symptoms might include, for example, distant vaginal erosion in a vaginal cul de sac or erosion of an adjacent organ (i.e., urethra, bladder, or rectum).

Type 3: Symptomatic Contractions

Implant retraction due to the retraction of tissue around the prosthesis is almost systematic and can reach up to 25–30% of the implant surface in rats, while in operated patients, it has been reported to reach up to 40% of the original surface. This is why many surgeons prefer to use a substantially sized implant to anticipate and cover the defects. Retraction need only be a source of concern either when there is poor coverage of defects, a source of recurrence, or clinical signs, especially pain. The description of the complications must first of all give the clinical signs presented by the patient, before the clinical extent of the retraction. These signs may consist simply of pain at palpation of the implant, during sexual relations or physical activities or, in extreme cases, permanent pain. The frequency (occasional, frequent, or permanent) and effects of these pains should be noted. It is sometimes difficult to judge to what extent the implant is the source of these pains, but it can be said to be responsible if palpation of the retracted implant produces the same pain as described by the patient. Palpation will also reveal the degree of retraction from under one-third of its initial surface to over two thirds.

In the most debilitating cases, if the implant is thought to be responsible, medical treatment should always be attempted, with antalgics, local anti-inflammatory injections, and local hormonotherapy. If the symptoms persist, the patients should be referred to specialist centers for a possible partial or total resection. Some of these complications may, of course, occur simultaneously.

Implant complications are often poorly managed or insufficiently evaluated, for aforementioned reasons. Ensuing treatment can vary widely, as there are still no clinical guidelines. It is easy to repair an implant exposition, when local trophic treatment has failed, through partial removal of the implant and an excision/suture of the vaginal scar. Conversely, the total removal of an implant near the bladder or rectum is a more delicate procedure. The removal, rarely necessary, of the prosthetic arms fixed in the obturator hole or in the sacrospinous ligament requires more experience. In very rare cases, augmentation vaginoplasty will be required. Serious complications such as these must be treated by skilled, experienced surgeons. DeLancy et al. will shortly be publishing a study of 13 complications requiring total or partial implant removal, referred between August 2005 and November 2007.[53] The authors rightly complain that they do not know how many prostheses were implanted in their area over the period in question and they are campaigning for a national register, which we are planning to set up in France. We have ourselves performed some 160 implant removals since 2000, the year when the use of vaginal prostheses began in France.

Pain and/or dyspareunia was the reason for removal in a quarter of the cases, mainly due to substantial retraction of the prosthesis. It is worth examining the incidence of dyspareunia, which is what most surgeons fear the most and which is why some of them will use vaginal implants only for women who are no longer sexually active. While until recently it could be acknowledged that the functional and especially sexual consequences of vaginal implants had not been sufficiently evaluated, several articles either recently published or to be published shortly, have examined the sexual consequences. Nieminem et al.[54] conducted a randomized study, in which they compared 99 anterior colporraphies (48 sexually active patients) with 105 supportive prostheses (49 sexually active patients): the dyspareunia score was statistically significantly lower ($p=0.015$) in the latter group! It could be argued that, in this study, the implant had no transobturator arm, which could be the reason for this excellent tolerance. Other studies have, however, supported the findings.

Lowman et al.[55] have studied the sexual outcomes of 129 Prolift implantations, three quarters of which were total Prolifts; the study comprised 57 sexually active women with a preoperative rate of dyspareunia of 36.8% and a postoperative rate of de novo dyspareunia of 16.7%. Of the 21 dyspareunic patients, less than a quarter considered the discomfort to be severe, while 75% would agree to undergo new surgery (83% for the cases of de novo dyspareunia).

Sentilhes et al.[56] had 37 sexually active patients out of their study of 83 patients fitted with implants (posterior IVS and anterior transobturator arms) investigated by complete sexual questionnaires (Lemack-Zimmern and PISQ 12). Results failed to show any difference between pre- and postoperative scores.

Gauruder-Burmester et al.[57] conducted a highly detailed, 12-month study of the sexual function of 120 women who had Apogee/Perigee mesh insertion: the operation was successful for the 15 women suffering from prolapse-related preoperative dyspareunia, while the sexual dysfunction that 40 women complained of was not secondary to the surgical intervention. The conclusion is that a prosthetic repair does not affect a healthy sex life!

Conclusion

In conclusion synthetic implants:

1. Must not be physically modified by tissue fluids
2. Must be chemically inert
3. Must not induce an inflammatory reaction or antibodies
4. Must not be carcinogenic
5. Must not induce allergy or hypersensitivity
6. Must be able to resist mechanical stress
7. Must be able to be manufactured in the required shape
8. Must be able to be sterilized

   Three criteria should also be added to this list:

9. Resistance to infection
10. Prevention of adhesion at the surface in contact with viscera and
11. Better in vivo response than autologous tissue

This review of existing prosthetic products demonstrates that no perfect product currently exists. Two categories of products seem to have promising properties with regard to their use in transvaginal surgery for restoring pelvic function. At one end of the spectrum, there are synthetic implants with mechanical properties of strength and elasticity, essentially made of polypropylene. Their strength is unchallenged, but whether they are well tolerated when introduced by the vaginal route remains uncertain. On the other end of the spectrum, there are animal collagen implants that appear to be well tolerated, but require clinical validation for this indication. Technical modalities for use are still undergoing validation and should allow a better understanding of the respective indications of these products in the correction of prolapse or urinary incontinence by the vaginal route.

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8

Medium Term Anatomical and Functional Results of Laparoscopic Sacrocolpopexy Using Xenografts

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Laparoscopic Sacrocolpopexy

Laparoscopy may yield better exposure and surgical detail, reduce blood loss and the need for excessive abdominal packing and bowel manipulation, which may all lead to a lesser morbidity.[1] Laparoscopy has now also found its way to the field of urogynecology. Recently, laparoscopic colposuspension was shown to be equally effective as an open procedure at 2-years follow-up.[2] Whereas colposuspension as primary therapy for urinary stress incontinence is on its way back, because of a lesser invasive and equally effective vaginal approach, other urogynecologic procedures may still benefit from an abdominal approach. Surgical repair of level I or apical vaginal defects, that also preserves vaginal function, can be performed either vaginally or through abdominal approach.[3] Randomized trials, however, have shown that sacrocolpopexy offers lower recurrence rates and less dyspareunia than sacrospinous fixation, but at the expense of a longer recovery time.[4] Logically, laparoscopic sacrocolpopexy (LSC) may reduce the latter morbidity. LSC was embraced later than colposuspension, probably because vault prolapse occurs more rarely and LSC needs extensive dissection and advanced suturing skills.[5] Data on LSC initially were limited to observational studies of variable size.[6–14] They covered issues such as perioperative parameters, reported short-term results, and were usually retrospective in design. In the largest retrospective study ($n = 363$) anatomical cure rate was 96% at a mean follow-up of 14.6 months.[10] Higgs observed on a longer term 8% recurrences at the level of the vault, but over one in three recurrences in the anterior or posterior compartment. The overall reoperation rate for prolapse was 16%.[11] We recently reported our prospective experience with all consecutive LSC beyond our learning curve. [15] LSC was introduced in our unit in 1996. Since, laparoscopy was used as the *primary* access route. In order to avoid an effect on outcome of the inherent learning process such procedure involves, we used the cumulative sum analysis (CUSUM) method to determine the learning curve.[16] Based on a 90% rate of avoiding *conversion* to laparotomy or occurrence of perioperative complications, our prior learning curve was set at 60 cases. Later cases (>61) were included in a prospective consecutive series of 132 women. They all had vaginal vault prolapse, defined as minimally presenting as Stage II apical prolapse. They underwent LSC using a Amid type I polypropylene implant over a 5-year period and were prospectively followed up by a standardized protocol to determine anatomical cure (≤Stage I per the pelvic organ quantification system [POP-Q])[17], subjective cure, and impact on quality of life, as measured by a standardized interview and a prolapse-specific questionnaire (P-QOL) before and after the operation.[15,18] The standardized interview consists of 28 questions related to prolapse, bladder, bowel, and sexual function. P-QOL assesses the impact of prolapse on nine different quality of life domains with scores for each domain, ranging between 0 and 100. Postoperative assessment was done after 3, 6, and 12 months and annually thereafter by a single independent assessor. De novo symptoms were defined as symptoms that were not present before surgery but present at the 3 months visit. At study closure all patients were asked to complete the P-QOL. If patients did not attend their planned follow-up visits, they were phoned to come for clinical assessment and if that was not possible, a telephone interview was undertaken to document the functional outcome. Primary outcome measures were anatomical and subjective cure. Anatomical cure was defined as the absence of Stage 2 prolapse or more at any anatomical site at any point in time during follow-up. Women reporting "never" or "rarely" reporting prolapse symptoms (questions 1, 2, or 3 of the standardized interview) were classified as subjectively cured.

Perioperative parameters and complications were comparable to the published literature (Table 8.1). We had comparably shorter operating times than what was reported in other laparoscopic series, but significantly longer than what is expected for open cases.[12,20] There were two early (<6 weeks) local infections, of which one required removal of an eroding

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Table 8.1 Perioperative characteristics and complications in a prospective series of 132 LSC. From Claerhout et al.[19]

	Mean	SE or %
Operation time (min)	180.5	(46)
Blood loss (mL)	185	(124)
Inpatient days (days)	5.7	(1.9)
Conversion	1	(0.7)
Complete laparoscopy	131	(99)
Preoperative complications	0	(0)
Complications in the early postoperative (<6 weeks) period		
Bleeding	1	(0.75)
Nerve lesions	3	(2.3)
Local problems	2	(1.5)
Late complications (any point in the follow up – range: 6–59 months)		
Reintervention related to the mesh	9	(6.8)
Mesh erosion	6	(4.5)
Pain related to mesh	3	(2.3)
Reintervention for genital prolapse	0	(0)

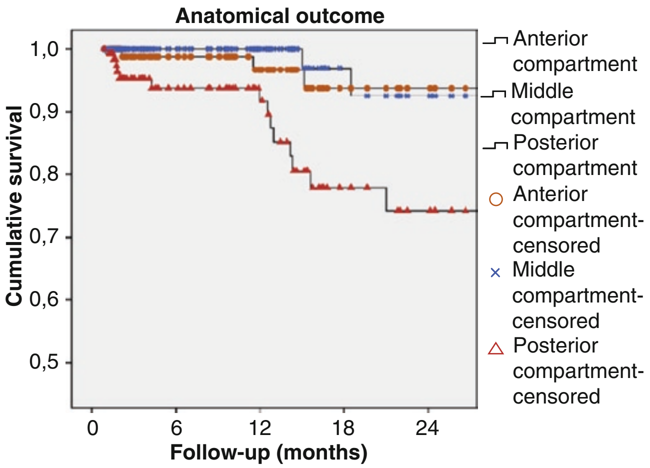

Fig. 8.1 Kaplan-Meier Survival curve for the different vaginal compartments from preoperative status (0 months) up to 24 months follow-up after LSC with synthetic grafts (Reprinted from Claerhout et al.[19] With permission)

mesh, without recurrent prolapse. At a mean follow-up of 12.5 months the anatomical level I (apical) cure rate was 98%. The overall anatomic cure rate was 94.7%, and all failures were confined to the anterior ($n = 1$) or posterior compartment ($n = 6$) (Fig. 8.1). Most patients (86%) defined as *anatomical* failures were asymptomatic. Subjective prolapse cure rate was 91.7%, occurring at a median of 17.4 ± 11.4 months (range 2.1–33). In only 30% of these, objective failure could be seen; none of the patients requested reoperation for recurrent prolapse. Symptoms of preoperative SI, urge incontinence, or constipation were cured in, respectively, 43%, 46%, and 42% of patients. The rate of de novo SI (i.e., patients with negative preoperative urodynamics) was 7.3%. De nove constipation developed in 5% and 21% de novo dyspareunia. Their quality of life improved significantly. Six erosions occurred in 4.5%, all within 1 year. All presented with vaginal discharge between 6 and 32 weeks postoperatively and required vaginal revision with partial mesh excision after failing local antibiotic and estrogen cream. This erosion rate is comparable to what other large studies report.[21]

Three additional patients underwent a vaginal revision because of dyspareunia that we related to folds in the mesh, all also eventually recovering. A significant improvement for all domains measured by the QOL-questionnaire was observed at 6 months and at study closure (Fig. 8.2). This largest, prospective, single center cohort study beyond the learning curve demonstrated that LSC also resulted in our hands in excellent anatomical outcome and subjective cure of prolapse symptoms at medium term (Table 8.2). The posterior compartment was most vulnerable for recurrence. Whereas sacrocolpopexy improved sexual function, still 25% at 6 months and 33% at study closure complained of vaginal symptoms interfering with sexual activity. This is higher than what is reported by Handa et al. (7.1% at 1 year).[22] We can only speculate about this apparent difference. Overall our patients were older, and there was a higher number of patients with preoperative impairment. On the other hand we do a very extensive lateral and downward dissection and lateral fixation, which may increase that risk.

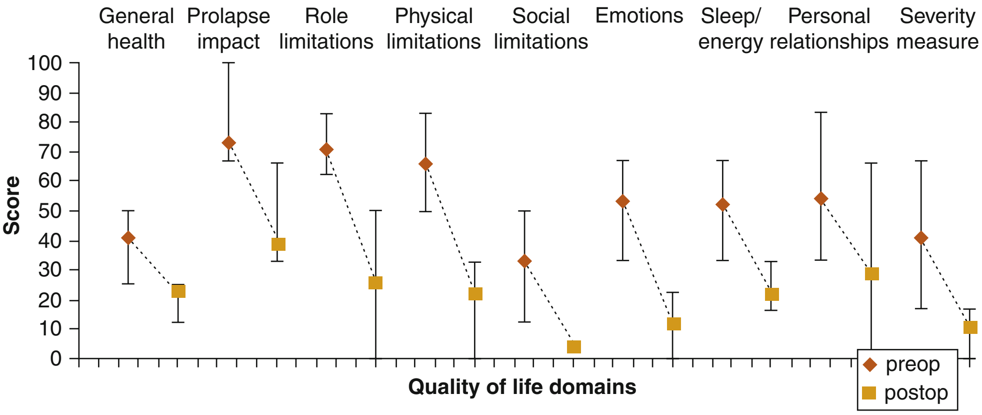

Fig. 8.2 Preoperative and postoperative (at 6 m) quality of life scores (P-QOL) expressed as mean and IQR (interquartile range) for patients completing preoperative and postoperative questionnaire (*n* = 36) after LSC with synthetic grafts (Reprinted from Claerhout et al.[19] With permission)

Table 8.2 Anatomical findings prior to, 3 months after surgery and at study closure in a prospective series of 132 LSC (Reprinted from Claerhout et al.[19] With permission

POP-Q	Prior to surgery		3 m		Study closure	
Number of patients at each time point	132		123		99	
≥ All compartments	132	(100)	7	(5.3)	22	(22)
POPQ point Ba ≥ −1	72	(54.5)	1	(0.8)	3	(3)
POPQ point C ≥ −1	87	(66)	0	(0)	2	(2)
POPQ point Bp ≥ −1	123	(94)	6	(4.5)	18	(18)

Rationale for Using a Xenograft Mesh

In view of the occurrence of local complications, in particular erosions and pain, the search for alternative implant materials has not ceased.[15,21,22] Permanent synthetics induce a strong and persistent inflammatory reaction, which may explain the durability of the repair but may cause graft related complications (GRCs).[23,24] Therefore, xenografts became considered because they provoke a milder inflammatory response.[24,25] This theoretically may lower the risk for local complications. For that reason we embarked on another clinical study, taking profit of the above standardized prospective follow-up scheme for patients undergoing LSC at our unit. We wanted to compare the outcome and complication rate following sacrocolpopexy with xenografts as compared to polypropylene. We hypothesized that xenografts would reduce the number of GRCs without compromising durability of the repair. Marketed heterologous porcine grafts in our country were at that time small intestinal submucosa derived from the pig (e.g., SURGISIS; Cook, Strombeek-Bever, Belgium), which is resorbable, and cross-linked dermal collagen (e.g., PELVICOL; Bard, Haasrode, Belgium).[24, 25, 26, 27] In the late 1990s, encouraged by clinical experience in hernia surgery, both products became approved by the FDA for use in pelvic floor surgery as well as they obtained CE-mark (Conformité Européenne – a label confirming that the product is fully compliant with European regulation).

Operative Technique

A single shot of intravenous cefazolin 2 g and metronidazole 1,500 mg was routinely given at least 1 h preoperatively. Patients were positioned in a modified lithotomy with access to the vagina and rectum. At least four cannulas were used: an umbilical 12 mm balloon cannula for open laparoscopy and two lateral 5 mm and one suprapubic 12 mm cannulas. With monopolar scissors, an area as large as required for fixing the mesh was dissected on the promontory, just right from the midline (Fig. 8.3a). The peritoneal incision was extended along the rectosigmoid to continue over the deepest part of the cul-de-sac, opening the recto- and vesicovaginal space. The lateral as well as the dissection downward toward the perineal body was extended as far as clinically required. Two separate meshes were sutured to the posterior and anterior aspect of the vagina using Ethibond 0 (Ethicon) sutures, with minimally one lower anterior, posterior, and apical row of three sutures (Figs. 8.3 and 8.4). The posterior mesh was also fixed laterally to the levator muscle. After that, the vault was first positioned at the level of the ischial spines, and then fixed tension-free and with three staples (EMS-endostapler, Ethicon, Groot-Bijgaarden, Belgium) to the promontory. This can obvioulsy be done with sutures as well. The meshes are then trimmed to the anatomical needs. The operative field was reperitonealized using a running Monocryl 0 (Ethicon)

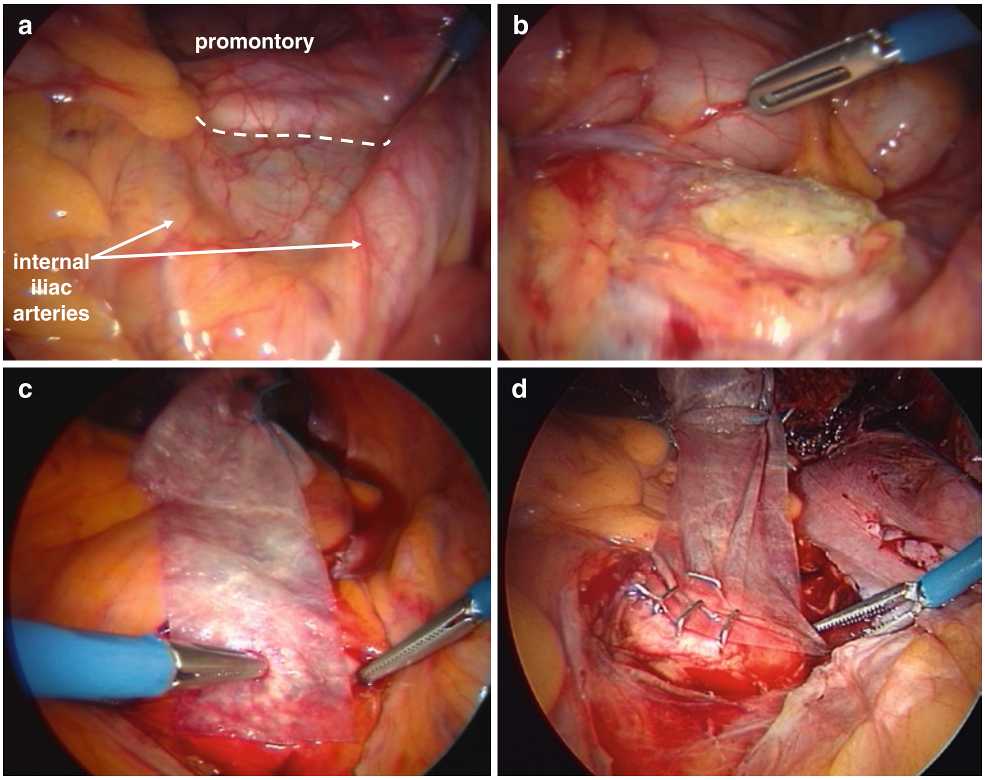

Fig. 8.3 (**a**) Anatomy of the promontory area, with the iliac arteries, and below the compressed large veins with a blueish shining. The dotted line is the inferior border of these vessels. (**b**) Status after dissection of the promontory. (**c**) SIS mesh is being sized to allow tension-free suspension of the vault. (**d**) SIS mesh stapled with EMS hernia staples to the promontory. SIS is more transparent than Pelvicol

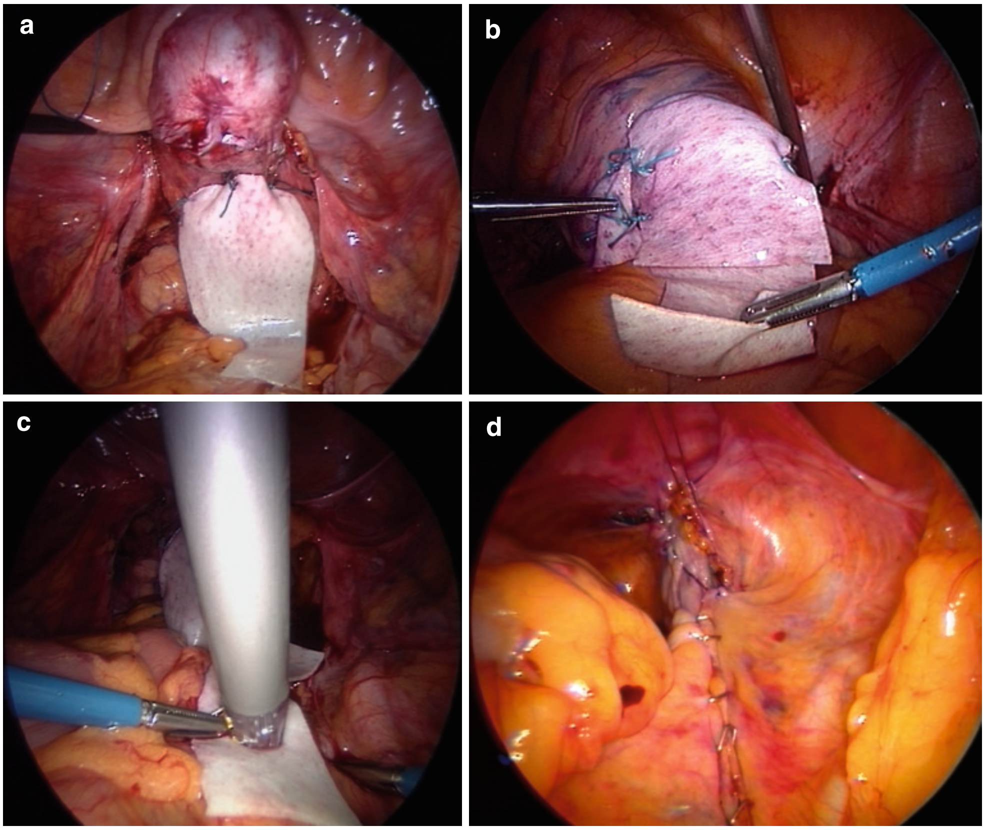

Fig. 8.4 (**a**) Pelvicol implant being sutured to the posterior aspect of the vault, with the three lower sutures already in place. (**b**) The anterior mesh as well as the posterior mesh are already sutured to the vault; the two implants are interconnected with Ethibond sutures. (**c**) Pelvicol mesh being stapled to the promontory. (**d**) Status after peritonealization with EMS staples (presacral area) and a running suture lower in the pelvis

suture. Prophylactic low molecular weight heparin injections as well as a stool softener macrogol 3.350 13.25 g (Movicol, Norgine) were continued for 6 postoperative weeks and sexual inactivity was requested until the 3-month visit.

Experience with Laparoscopic Sacrocolpopexy Using Xenograft

This was again a prospective observational study including 150 consecutive patients scheduled for laparoscopic sacrocolpopexy (LSC). In one consecutive cohort of 50 patients we substituted the polypropylene graft by a porcine-derived xenograft. We empirically choose this sample size as no clinical data were available at that moment. Half of these would have a Surgisis implant, the other half Pelvicol, again consecutive patients. We had no aim to compare outcomes between the two xenografts. The parameters, study design, and surgeon were identical to what was described above. Post hoc, we choose a double sized, unselected control group of 100 patients operated with a synthetic polypropylene graft. We based our sample size such as to detect a 25% difference in anatomical cure rate between patients operated with xenogenic and synthetics grafts. Based on observations in the study group, and an expected anatomical cure rate of 78% in patients operated with PP, we calculated that a control group of at least 65 patients and a treatment group of 35 patients would permit to demonstrate a 25% difference (two-sided test, $\alpha = 0.05$ and power = 0.8). To allow a comparable duration of follow-up in both groups, we selected 50 consecutive controls operated immediately before, and 50 immediately after the xenograft-cohort.

There were no differences, apart from age, between the groups (data not shown).[28] The mean follow-up in the xenograft and control group was 32.6 months (range: 20–68) and 33.5 months (range: 6–93) (not significant),respectively. Overall anatomical failure rate was comparable (49% vs 34%; $p = 0.053$), but failures at the vault (21% vs 3%; $p < 0.01$) and posterior compartment (36% vs 19%; $p < 0.05$) were more frequent in the xenograft group (Table 8.3). The time point to first presentation of recurrence for any compartment is displayed in Fig. 8.5. The median time to anatomical failure in the xenograft group was 30 months (range: 1–84) as compared to 15 months in the control group (range: 2–69) ($p = 0.14$). More than 60% of all patients identified as *anatomical* failures were asymptomatic without differences between groups (xenografts: 53% vs controls: 73%; $p = 0.14$). There were six reoperations for prolapse, all confined to the xenograft group ($p < 0.01$). Five of them underwent a secondary LSC with PP (range: 12–60 months after initial surgery) and one a vaginal cystocele repair at 48 months (Fig. 8.6).

There were 12(12%) GRCs and these were equally frequent in the xenograft and controls (Table 8.4). Most common were erosions ($n = 8$). Two occurred in the Pelvicol operated patients: one presented as early as 8 weeks postoperatively with discharge and mesh exposure, leading eventually to revision after 15 months. The other patient presented with vaginal bleeding 4 years postoperatively, and implant material was visible next to granulation tissue.

Table 8.3 Anatomical failures at different compartments and GRC by graft material as well as the number of reinterventions for prolapse and GRC evaluated in 150 patients undergoing LSC either with a xenograft or polypropylene (n(%)) (From Deprest et al.[28])

Number of patients available for clinical evaluation	Xenografts			Controls operated with poly-propylene	p-Value[a] controls versus *xenografts*
	SIS (n = 18)	Pelvicol (n = 21)	All xenografts (n = 39)	n = 65	
≥Stage 2 all compartments	11(61)	8(40)	19(48.7)	22(33.8)	0.053
POPQ point C ≥ −1	4(22)	4(19)	8(20.5)	2(3.1)	0.004
POPQ point Ba ≥ −1	4(22)	6(29)	10(25.6)	9(14.1)	0.132
POPQ point Bp ≥ −1	7(38.9)	7(33)	14(35.8)	12(18.5)	0.047
Reoperations for prolapse					
Vault prolapse	2(9.5)	3(14.2)	5(12.8)	0	0.006
Anterior compartment	0(0)	1(4.7)	1(2.6)	0	0.375
Posterior compartment	0(0)	0(0)	0(0)	0	1
Graft-related complications					
All GRC	2(11)	2(9.5)	4(10.2)	8(12.3)	0.635
Local infection	2(11)	0(0)	2(5.1)	0(0)	0.044
Implant exposure	0(0)	2(9.5)	2(5.1)	6(9.2)	0.607
Pain requiring graft revision	0(0)	0(0)	0(0)	2(3.1)	0.412
Reoperation for GRC	0(0)	1(4.8)	1(2.6)	7(10.8)	0.199

[a] Calculated with Pearson chi-square test

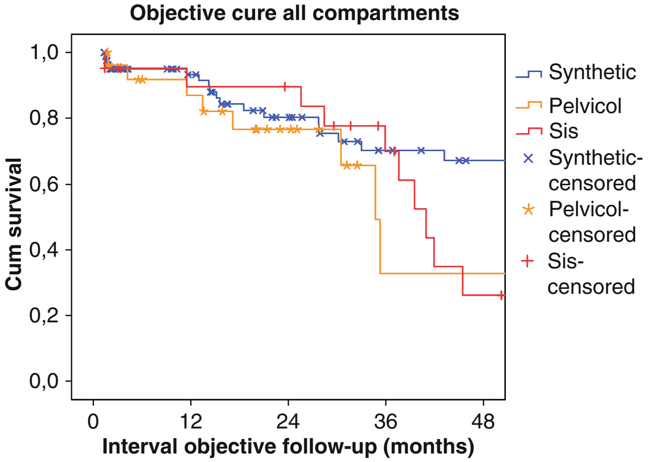

Fig. 8.5 Kaplan-Meier Survival curve for the different vaginal compartments from preoperative status (0 months) up to 24 months follow-up after LSC with either xenogenic or synthetic grafts (From Deprest et al.[28])

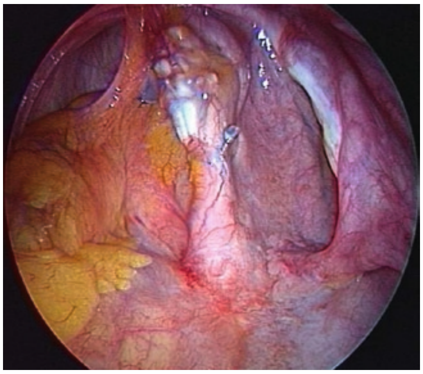

Fig. 8.6 Status 9 months after sacrocolpopexy with a Pelvicol implant. The patient underwent laparoscopy for unrelated reasons

The symptoms disappeared following local clindamycin and estriol. The other six presented with obvious erosion over the polypropylene mesh between 6 and 32 postoperative weeks. All except one required vaginal revision. Two additional patients from the polypropylene-control group underwent vaginal revision because of palpable folding of the mesh coinciding with dyspareunia. There were two vaginal vault infections, both in the SURGISIS group. One of them presented 9 weeks postoperatively as a pelvic abscess that drained spontaneously. The implant material could easily be removed in the office, and this patient did not develop recurrence (follow-up: 50 months). The other patient presented 6 months postoperatively, with painful swelling and signs of inflammation on the vaginal vault, but without abcedation or fever. All signs disappeared under oral antibiotics. Overall, GRCs were equally frequent (11%) in the xenograft and polypropylene group. The reoperation- rate for GRCs was not different (xenograft 3% vs controls 11%; $p = 0.20$).

The pelvic floor symptoms reported at baseline and at study closure are displayed in Table 8.4. The effects of LSC on pelvic floor function were similar for both groups. The percentage of patients with de novo stress incontinence, urge incontinence, constipation or dyspareunia in the xenograft group was 0%, 4%, 2%, and 4% and 7%, 6%, 9%, and 10% in the control group, respectively, all differences not being significant. We concluded that sacrocolpopexy using xenograft was associated with more apical failures and reoperations for prolapse than when using PP, without difference in functional outcome. The use of xenografts did neither reduce GRCs.

Discussion

In our experience, we observed a significantly higher apical failure and reoperation rate (15%) for prolapse following LSC using xenografts, without reduction of the number of GRCs. The functional outcomes were not different. This is obviously only one experience, and the study design was far from ideal. However, this was a prospective study without selection of patients operated with xenografts based on patient characteristics. Also the study pools the observations on xenografts, for two materials that are dramatically different. SURGISIS is a resorbable product, whereas PELVICOL is cross-linked. Recent other studies on sacrocolpopexy using xenografts reported, however, an equal or even higher failure rate. Altman et al. documented no difference in anatomical failure rate (≥Stage II point C) between patients operated with PELVICOL ($n = 27$) or synthetic mesh ($n =2\ 5$) (29% vs 24%; mean follow-up: 7 months).[29] Quiroz et al. compared the anatomical outcome and GRCs of 134 synthetic, 102 PELVICOL, and 23 autologous fascia sacrocolpopexies.[30] Also in those studies, patients were not consecutive, but rather chosen on patient characteristics. After a mean follow-up of 1.2 years Quiroz et al. observed 11% apical failures at a median of 9 months in the PELVICOL group (however defined as > Stage 0 or reoperation for recurrent apical prolapse), whereas recurrence was 1% for synthetics. With a less stringent definition (>Stage I) and a median follow-up of 34 months, we observed 19% apical failures following PELVICOL LSC, as opposed to 3% for controls. Our median time to recurrence in the PELVICOL group was 2.5 years and >75% of the recurrences presented beyond 12 months. Our longer follow-up period may be partly responsible for an apparent higher failure rate, and underscores the importance of a sufficiently long follow-up. Experimental studies have demonstrated that non-cross-linked grafts are prone to rapid degradation and reherniation.[25,31] Cross-linked materials such as PELVICOL are rather encapsulated, hence less integrated. They may also be affected by late degradation due to a foreign body reaction.[32] A study on the histopathologic detail found in these individuals has shown that recurrence coincides with local degradation. We also observed that the GRC-rate was equally high in both groups (11–12%). Quiroz et al. observed an even higher

Table 8.4 Urogenital symptoms prior and at study closure, as evaluated by standardized interview in 150 patients undergoing LSC either with a xenograft or polypropylene (*n*(%)) (From Deprest et al.[28])

	Preoperative				At study closure			
	Xenografts		Controls	*p*-Value[a] controls versus xenografts	Xenografts		Controls	*p*-Value[a] controls versus xenografts
	SIS	Pelvicol			SIS	Pelvicol		
Number of patients available for standardized interview	($n = 21$)	($n = 29$)	($n = 100$)		($n = 20$)	($n = 27$)	($n = 82$)	
Prolapse symptoms								
Prolapse symptoms	21(100)	29(100)	100(100)	1	5(25)	7(26)	11(13.4)	0.223
Urinary symptoms								
Stress incontinence	5(24)	6(21)	22(22)	0.966	3(15)	3(11)	18(22)	0.411
Urge incontinence	3(14)	6(21)	24(24)	0.609	3(15)	5(18.5)	12(14.3)	0.888
Urgency	5(24)	8(28)	32(32)	0.721	6(30)	6(22)	14(17)	0.415
Defecation symptoms								
Constipation	9(43)	2(7)	27(27)	0.012	7(35)	2(7)	19(23)	0.066
Fecal incontinence	3(14)	3(10)	9(9)	0.762	1(5)	2(7.4)	9(12.2)	0.241
Digital support	6(29)	2(7)	16(16)	0.119	4(20)	2(7)	11(13)	0.306
Sexual symptoms								
Sexual activity	13(62)	15(52)	59(59)	0.706	8(40)	12(44)	39(48)	0.788
Dyspareunia	2(15)	3(20)	9(15)	0.903	3(37)	3(8)	12(31)	0.527

[a] Calculated with Pearson chi-square test

GRC rate with PELVICOL instead of synthetics. In both studies the overall reintervention rate for GRC was not significantly different between xenografts and synthetics.[30]

We tried to draw some clinical conclusions from our observations. The lower anatomical cure rate prompts for cautious use of xenografts for sacrocolpopexy. Meanwhile, we resorted again to PP-grafts for sacrocolpopexy, and reserve xenografts to patients at high risk for local complications on PP-grafts, although the efficacy of that policy remains to be demonstrated. The search for better grafts should go on, as the ideal mesh has certainly not been identified, given that GRCs persist in both groups. The above clinical experience provides also proof that novel grafts should be tested first preclinically, followed by evaluation in properly designed observational studies.[33]

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Free or Fixed Implants?

9

Renaud de Tayrac and Pascal Mourtialon

Introduction

Pelvic organ prolapse repair is one of the most common operations in postmenopausal women. Abdominal reconstructive surgery, such as abdominal sacrocolpopexy, is classically more successful than vaginal repair.[1] The difference is probably less related to the way of approach than to the use of prosthetic materials for abdominal repairs. However, abdominal sacrocolpopexy exposes women to the risk of bowel obstruction, pelvic infection, and spondylodiscitis. Moreover, vaginal erosions are commonly reported in all series of sacrocolpopexy with an overall rate of 3.4%[2] and when vaginal erosion occurs, mesh removal by laparotomy could be necessary.

The vaginal route is preferred in many conditions, such as stress urinary incontinence, hysterectomy for benign disease, and post-hysterectomy vaginal vault prolapse. Reasons are simplicity, reproducibility, low morbidity, and decreased postoperative pain, hospital stay and cost. Unfortunately, most of procedures using conventional techniques[3,4] or absorbable meshes[5,6] for prolapse repair by the vaginal route result in an unacceptably high recurrence rate, up to 50%.

The use of a nonabsorbable prosthetic mesh in vaginal surgery was first introduced by Julian in 1996[7], who showed in a randomized study a significant reduction of recurrence rate when a polypropylene mesh was used as tissue support. During the last 10 years, many case series of vaginal surgery using polypropylene meshes have been reported, with promising anatomical success rates, ranging from 75% to 100%, at short- to medium-term. Recently, three randomized controlled trials have confirmed the superiority of the use of material in vaginal reconstructive surgery rather than conventional techniques.[8-10] However, the way to insert the mesh is still unclear for both anterior and posterior repairs.

Our goal is to report the different techniques that have been studied and to compare respective outcomes.

Anatomical Considerations

Knowledge of anatomy is required for any surgery, but especially for vaginal surgery because some gestures are blind and the position of the patient on the surgical table change anatomical landmarks position studied in reference anatomical position.

The most important anatomical landmarks for the vaginal surgeon are:

- Retropubic space between pubic bone and bladder
- Paravesical space between bladder and pelvic side wall
- Arcus tendineus fasciae pelvis (ATFP) from ischial spine to ischio-pubic ramus
- Ischial spine
- Sacrospinous ligament between sacrum and ischial spine
- Ischioanal fossae
- Pararectal space between rectum and pelvic side wall

Three new surgical approaches have been described in the last 10 years for the use of mesh in vaginal reconstructive surgery:

1. *The* (*Anterior*) *Transobturator Route*[11]: Skin entry point is in the genitofemoral fold at the level of the cliroris and the internal point is in the paravesical space on the caudal portion of the ATFP. The path from superficial to deep is: gracilis muscle, adductor magnus muscle, obturator externus muscle, obturator membrane, and obturator internus muscle.
2. *The Posterior Transobturator Route*[12]: Skin entry point is in the genitofemoral fold 1 cm lateral and 2 cm below the previous incision. The path from superficial to deep is: adductor magnus muscle, obturator externus muscle, obturator membrane, and obturator internus muscle at the end on the ATFP 1cm distal to the ischial spine.
3. *The Ischioanal Route*[13]: Skin entry point on the buttock is 3 cm lateral and 3 cm posterior to the anal verge. The route passes through the ischiorectal fossae to enter the pararectal space. The end is in the iliococcygeus muscle or through the sacrospinous ligament 2 cm medial to the ischial spine.

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Experimental Considerations About Free or Fixed Implants

Tension-free techniques for surgical prolapse repair with mesh implanted vaginally were derived from the reliable concept of the tension-free vaginal tape.[14] It consists in the passage of the mesh into tissues without dissection. The mesh is maintained in place by interactions between living tissues and the material used. This “Velcro” effect or “seal skin” adhesion were used to explain the tissue maintenance, until the strengthening of the device by healing.[15] However, few studies have been performed to confirm that hypothesis experimentally.

The healing process, tissue adherence and fibroblastic colonization of tension-free devices, depend on mesh dimensions.[16-18] While waiting for this scarring process, the tissue maintenance can only resist coughing and other abdominal pressure in the immediate postoperative period. Meshes used for pelvic floor repair are subjected to great forces due to a large surface in contact with the prolapsed organs. Boukerrou et al. have shown that an increase in the width of the tapes of 0.5 or 1 cm involves a better tissue resistance, probably due to a larger surface area in contact with tissues.[15] The relation between surface and mechanical properties is probably not linear but exponential with the release of the arms of the mesh. In the same way, the trans-sacrospinous ligament route should be preferred to the transmuscular one when using wider tapes. It is preferable to use a tape of at least 2 cm through the sacrospinous ligament, which has a much better fixation, especially for the treatment of prolapse where the forces are larger due to an increased contact surface area.[15]

Cosson et al. have studied the resistance until rupture of ligaments in reconstructive pelvic surgery, for colposuspension, paravaginal repair, sacrospinofixation or sacrocolpopexy and observed that resistance was highly variable, from 20 to 200 N[19]. The difference in tissue resistance between tension-free procedures and classic operations is difficult to explain, but Boukerrou et al.[15] have proposed a physical theory. In the Burch colposuspension, forces are distributed at four points, on vaginal sutures and pectineal ligaments (Fig. 9.1a). If the restraint passes the strength limit, one or more of the fixation points may release. This can explain the irreversible rupture during great pressure in the postoperative period. On the contrary, in suburethral sling procedures, the forces are distributed at many points along the contact surface with the living tissues (Fig. 9.1b). Hence, during stress in the immediate postoperative period, the tape follows the movement of the nearby tissues. If there is a mobilization of some fixation points, the multiplicity of those points guarantees the device fixation. It is probably due to the elasticity characteristic of slings. In case of slings, the multiple fixation points due to the network and sutures of the material explain the good results obtained despite weak forces of rupture. If clinical results of tension-free procedures are of good quality, fixation mechanisms should be further studied. The study by Boukerrou et al.[15] was the first approach to tension-free tissue strength. At the end of their work, they have proposed the following recommendations:

1. The posterior arms of the mesh used for pelvic floor repair should measure at least 1 cm in width in order to improve postoperative resistance.
2. To improve suspension of the posterior arms in the transperineal mesh, passage through the sacrospinous ligament should be preferred to the transmuscular route.
3. The network of the meshes influences tissue resistance and therefore specific tension-free materials must be studied before being made commercially available by the manufacturer.

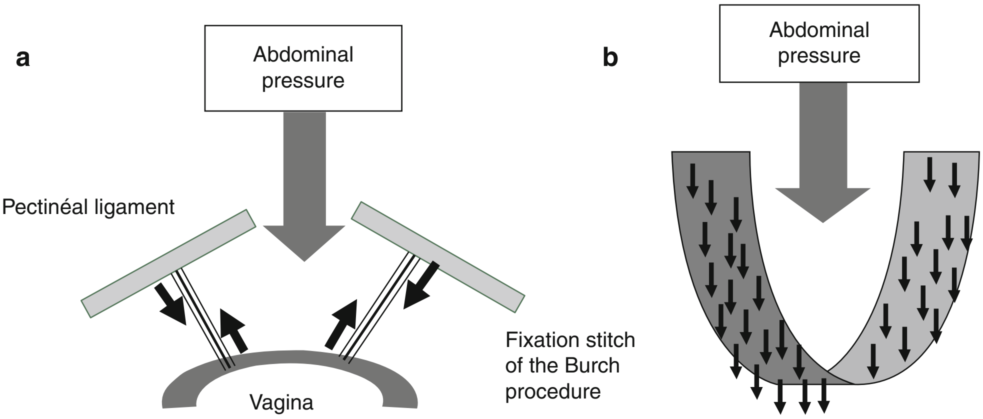

Fig. 9.1 Difference in tissue resistance between colposuspension (**a**) and tension-free vaginal tape procedures for surgical treatment of stress urinary incontinence. (**a**) Distribution of forces after Burch colposuspension. Arrow denotes restraints forces on the fixation points due to abdominal pressure. (**b**) Distribution of forces after tension-free vaginal tape. Arrow denotes distribution of abdominal forces restraints forces on the surface of the tape (Reprinted from Bourkerrou M et al.[15] With permission from Elsevier)

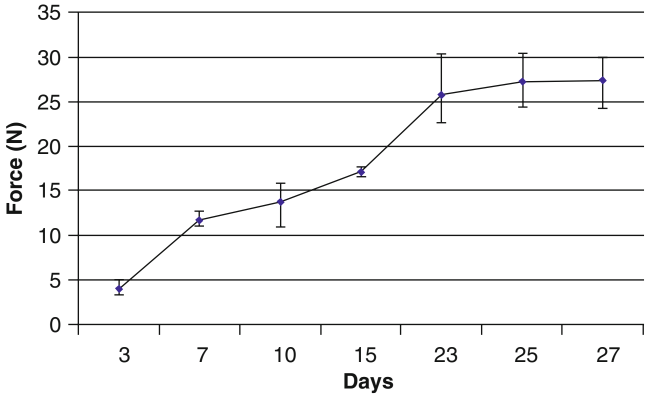

Fig. 9.2 Relation between force and time to healing. Resistance gain (in Newtons) of pull-out forces needed to mobilize meshes (in days) (From Boukerrou M et al.[20] With permission from Elsevier)

In another experimental protocol in the back wall of rats, Boukerrou et al.[20] have shown a linear relation between tissue resistance and delay in cicatrization until about day 25 (Fig. 9.2). The curve plotting the force of tearing against resistance shows a plateau at about 27 N. Continuing measurements until day 60 demonstrate that this plateau is then stable, the necessary force needed to tear the mesh from tissues being around 27 N at that stage. Around day 25 after surgery, they observe the maximal resistance of montages given by cicatrization. Neoformed connective tissue allows incorporation and tissue integration, increasing the "seal-skin" effect initially described and measured immediately postoperatively. They also observe that, when the local infection develop around the mesh, cohesion forces are weak, never above 6 N, no matter how many days of cicatrization. Resistance is homogeneous, lower than normal resistance in cicatrization without infection at day 7. Low resistance to traction any time after surgery does correspond with the clinical observation of patients where the mesh could easily be removed by simple traction. Inflammatory shell and pus prevent any tissue fixation and block any adapted incorporation of the mesh into normal scarring tissue.

Rat is often chosen as an immunologic model concerning the evolution of cicatrization. From studies in immunology and plastic surgery, we know that cicatrization in the rat can be extrapolated to humans.[21,22] Fibroblasts and neoformed connective tissue steal their way through polypropylene fibers, organize in the net, and mature with the help of neovessels. Collagen appears and organizes itself to include the mesh and reinforce the mesh tissue cohesion.[23] The kinetics of this inclusion showed, in the rat model, maximal reinforcement around the 25th day. This schedule cannot be directly extrapolated to human, but healing kinetics is very nearly identical. An increase in resistance to traction is linked to cicatrization, mainly collagen fibers invading pores of the mesh. These observations support the time when patients are usually asked to no longer require taking precautions after tension-free slings, 4–6 weeks, with minimal exercise and no handling of heavy weights, sexual intercourse, or sports. In N/mm^2, resistance to tearing increased from 4 to 27 N/300 mm^2 (0.013–0.09 N/mm^2) on days 3–25. Maximal resistance to coughing or abdominal pressure was found at 185 and 467 mmHg and at 7–18 N, respectively.[24] This resistance is linked to the maximal constraints of prosthetic meshes. Therefore, postsurgical safety time could be reduced to 2 or 3 weeks for physical strain or carrying of heavy weights. Another study by Rezapour et al. has been published on this subject.[25] The objective was to evaluate in a sheep model the effect of healing on mechanical properties. In that study, authors have added a composite of PDS® and Vicryl® to the end of the tape and tested the quality of friction along cicatrization. They confirmed that healing increased tissue ingrowth of mesh and the pull-out forces needed to remove the tape from tissues increase from 1 to 12 weeks.

Other factors that influence resistance are the physical characteristics of the mesh and size of the surface in contact with the recipient tissue.[26,27] It has been shown that the mesh, which has the best mechanical performance and the best tissue integration is the macroporous monofilament polypropylene.[28] Better early tissue incorporation (higher collagen deposition, capillarity density, and cell accumulation) increases the tensile strength, reflecting tighter anchorage to surrounding tissue.[29] Furthermore, in a swine model using abdominal wall implantation, Gonzalez et al. have shown a significant correlation between tissue ingrowth force and mesh size (p = 0.03, 95% CI: 0.05–0.84).[30] The more was the mesh size, more high was the tissue ingrowth force ($r = 0.5491$). The hypothesis is that mesh fixation with sutures holds the mesh in place while integration occurs, which could decrease risks of migration and contraction.[30] On the opposite, an increased inflammatory reaction resulting from an infection around the mesh could alter the tissue integration process and therefore provide inaccurate information.

In women, Tunn et al. have observed a high rate of 6-weeks postoperative mesh contraction, evaluating sonographically, after transobturator and transischioanal tension-free implants for anterior and posterior repair, respectively (from 6.8 ± 1.1 to 2.9 ± 0.6 cm and from 9.9 ± 0.8 to 3.3 ± 0.5 cm).[31] However, these authors used a transischioanal trans-coccygeus muscle posteriorly, instead of transischioanal trans-sacrospinous ligament that could provide less stability of the mesh. Furthermore, they did not investigate whether the mesh length was affected by the fact that no additional fixation stitches were placed.

Surgical Techniques

Concerning the use of prosthetic material in vaginal reconstructive surgery, aseptic rules are very important and common to all techniques:

- Antiseptic shower the day before surgery
- Shaving or shearing pubic hair and vulva
- Large brush of antiseptic solution before installation
- Isolation of the anus
- Careful mesh handling
- Changing gloves before any mesh handling
- Intraoperative antibiotic prophylaxis

A proper installation of the patient on the operating table is also important for the reproducibility of the intervention, since anatomical landmarks are changing dramatically with the position. Furthermore nerve damage has been reported due to poor installation.[32,33]

Preoperative local estrogen is usual for postmenopausal patients in order to facilitate scar tissue formation around the mesh.

Anterior Compartment

Julian first reported in 1996 the use of a prosthetic reinforcement cystocele repair with a Marlex mesh.[7] The anterior vaginal segment was reinforced by sewing the synthetic nonabsorbable mesh from the urethra-vesical junction anteriorly to the vaginal apex posteriorly and to the junction of the obturator internus and levator ani fascia at the lateral margins of this space. This mesh was placed after Pereyra urethropexy, anterior colporrhaphy, and bilateral transvaginal and paravaginal defect repair to restore the anterior vaginal segment. In order to prevent mesh erosion, vaginal flaps created during dissection of the anterior segment and close one over the other were used.

After that first description, many techniques of anterior repair with mesh have been described. Several surgical steps are common to most of these techniques. Almost all surgeons perform bladder dissection on an emptied bladder after implantation of a Foley catheter. Some operators infiltrate the vaginal wall by saline with a vasoconstrictive solution to facilitate dissection and reduce bleeding. A sagittal colpotomy is usually performed starting 2 cm away from the vaginal vault or uterine cervix and ending approximately 2–3 cm from the urethral meatus. That incision allows a separation between anterior repair and suburethral tape placement. A transversal incision at 2 cm away from the vaginal vault or uterine cervix is also feasible. The bladder is usually dissected laterally while keeping the pubocervical fascia on the vaginal wall, in order to increases mesh tolerance and tends to decrease the erosion rate. Many surgeons perform a plication of the pubovesical fascia before mesh placement. At the end of the procedure (after mesh implantation), the vagina is packed with a strip of disinfecting gaze for 24–48 h to avoid hematomas and to press the mesh tightly against the vaginal wall.

Free Implants

Real Free Implants

That technique of mesh placement was first described by Dwyer et al.,[34] also used by others[35-40] and employed in a large randomized trial.[8] The dissection is extended bilaterally to the ischial spines and advanced anteriorly along the ATFP. Midline plication of the fascial layer is performed using interrupted 2/0 absorbable sutures. The mesh is widely spread after opening the paravesical fossae and identification of the ATFP up to the ischial spine. Lateral extensions of the mesh are positioned onto the iliococcygeal fascia anterior to the ischial spines. The mesh is usually unsutured, although in cases of complete vaginal eversion, aborbable sutures could be placed into the iliococcygeal fascia and/or at the anterior and posterior margins for stabilization and to prevent folding.

In the Hiltunen et al. description[8], the dissection was more limited and the mesh had four arms inserted in four tunnels created by sharp and blunt dissection along the inside of the inferior rami of the pubic bone anteriorly, toward the obturator foramen but not reaching through the obturator membrane, and toward the ischial spine posteriorly. Efforts were made to keep the tunnels narrow enough to fix the arms of the mesh in place.

Retropubic Free Implants

That technique of mesh placement was first described by Zargar et al.[41], and also used by others.[42-46] After cystocele dissection, endopelvic fascia in either side of the bladder neck is perforated to enter into the retropubic space with the finger. Two anterior arms of the mesh are then entered into the retropubic space, without fixation, the body of the mesh staying free under the bladder.

A 3 cm skin incision above the symphysis pubis on abdominal wall has also been used, in order to perforate the rectus fascia. Using a nonabosrbable suture material, urethropelvic ligaments, vesicopelvic fascia, and cardinal ligaments could be grasped helically and separately using a Raz method[47], all are brought out onto abdominal wall over the rectus fascia (suspension). After that kind of suspension, cystoscopy has to be performed to detect any bladder injury.

Transobturator Free Implants

That technique of mesh placement was first described by Eglin et al.[48], using the anterior transobturator route from Delorme[11] and an original inferior transobturator passage for the posterior arm (Fig. 9.3). The transobturator technique is currently the most widely used[48-56] and two randomized trials have already been published.[9,10]

That technique was extensively developed by Jacquetin and eight other French surgeons, allowing the marketed of a specific kit.[49] After opening the paravesical fossa, ATFP is identified by palpation, from the posterior part of the pubic ramus to the ischial spine. Four skin incisions are made on the genitocrural fold: two incisions in the anteromedial edge of the obturator foramen at the level of the urethra and the two other incisions 2 cm below and 1 cm lateral to the first ones. Bilateral passage of the two upper cannula-equipped guides at 1–2 cm of the pre-pubic part of the ATFP and bilateral passage of the two lower cannula-equipped guides at 1–2 cm of the distal part of the ATFP (1 cm from the ischial spine) allows catching each prosthetic arm and passing them through the obturator foramen. Afterward, the mesh is positioned tension-free under the bladder. The mesh is sutured to the uterine isthmus (or to the vault) with a single stitch of nonabsorbable suture to provide apical suspension of the mesh.

Fixed Implants

Fixation to the Arcus Tendineus Fascia Pelvis

That technique of mesh placement was first described by Hung et al.[57], and used by other several authors.[58-60] The procedure consisted of an extensive vaginal dissection to join the vesicovaginal and retropubic space and anchoring of a polypropylene mesh between the two ATFP. Laterally, the epithelium is dissected to the pelvic sidewall from immediately behind the pubic ramus up to the level of the ischial spine. Careful examination in the area of the lateral dissection confirmed bilateral or unilateral paravaginal defects in all of the patients. These defects are characterized by partial or complete detachment of the pubocervical fascia from the ATFP. Blunt finger dissection is then used to gain complete access to the retropubic space bilaterally. The obturator internus fascia and the ATFP are identified by palpation and then visually. Two nonabsorbable sutures are placed in the ATFP in a helical fashion at the level of the bladder neck and just anterior to the ischial spine, respectively. The stability of the four sutures, which are anchored at the four corners of the bilateral ATFP, is tested before they are left untied and held with Kelly clamps. A midline plication of the pubocervical fascia is performed at this time. The previously prepared polypropylene mesh is further tailored to fit in the space between the four sutures and then the four sutures are passed through the lateral edges of the polypropylene mesh and tied. Care is taken to avoid exerting under tension on the bladder base by the polypropylene mesh. The anterior and posterior edges of the polypropylene mesh patch are further fixed at the level of the bladder neck and the cardinal ligament, respectively, with two lateral absorbable sutures. After a cystoscopy, vaginal flaps are trimmed and sutured with absorbable sutures. Fixation to the ATFP near the ischial spine in not very easy, that is why some use specific devices to perform the stitches.

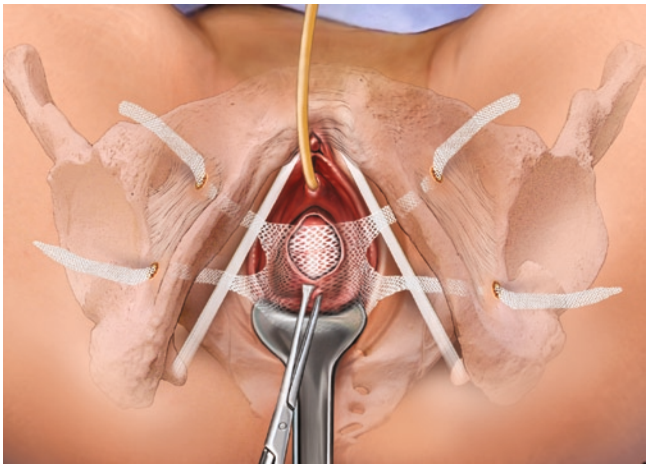

Fig. 9.3 Anterior repair with a transobturator kit (Reproduced from CR Bard Inc. With kind permission)

Fixation to the Sacrospinous Ligament

That technique of mesh placement was described and used only by Amrute et al.[61] They describe an anterior repair with an “H” mesh under the bladder; the two anterior arms are fixed to a retropubic tape and the two posterior arms to the sacrospinous ligament with the Capio suture-capturing device (Boston Scientifc Corp., Natick, MA, USA). After identification by palpation, two delayed absorbable sutures are bilaterally placed 1 cm medial to the ischial spines onto the sacrospinous ligaments using the Capio device. The bladder is sharply dissected from the vaginal mucosa and endopelvic fascia is imbricate with absorbable suture if necessary. Sharp dissection of the periurethral space is carried superiorly toward the retropubic space bilaterally. Based on patient anatomy and extent of the site-specific defect, a rectangular polypropylene mesh is fashioned into an “H” shape and the anterior arms are sutured to two polypropylene mesh arms. Using a tape needle passers, the anterior arms are passed retropubically exiting from the anterior abdominal wall. The distal edge of the mesh is positioned at the mid-urethra.

Cystoscopy is performed to evaluate any bladder perforations and to place a suprapubic catheter. Ureteral patency is also noted by excretion of indigo carmine dye administered intravenously. The sacrospinous ligament sutures are passed through the pores of the lateral edges of the posterior arms and the arms are tied down to the ligaments. The sutures are brought out at each side through the intact posterolateral aspect of the vaginal mucosa. Several absorbable sutures are placed to prevent folding and kinking of the mesh. The sacrospinous ligament sutures are tied down to provide apical support, along with posterior arms of the mesh and the sutures are transected flush against the vaginal mucosa. Ultimately, the mid-portion of the "H" mesh corrects the anterior middle compartment defect while the anterior arms create mid-urethral support.

Posterior Compartment

Several different techniques have also been described for posterior repair. Common steps are the sagittal midline incision from the vaginal vault or the posterior vaginal fornix, 2–3 cm above the uterine cervix to the perineal boby, rectocele, and/or enterocele dissection allowing opening pararectal spaces from the levator ani to the ischial spine and then to the sacrospinous ligament.

Free Implants

Real Free Implants

That technique of mesh placement was first described by Milani et al.[62], and also used by others.[38,40,46] The surgical procedure involves a conventional posterior repair (fascial placation) plus the placement of a mesh. After a midline incision, the vaginal wall is carefully dissected laterally, with the dissection made just beneath the vaginal mucosa, so that all fascial tissue is left attached to the rectum. The dissection is performed to the rectal pillars. The fascial tissue is then plicate with interrupted absorbable sutures. Transverse and anteroposterior dimension of the prolapsed segment is measured with a sterile ruler. This procedure determines the size of the mesh to be applied. Once inserted, the mesh is only fixed using absorbable sutures.

Low Transobturator Free Implants

That technique of mesh placement was described and only used by Sentilhes et al.[63] The authors describe a Y shape mesh that is sutured posteriorly to a posterior transischioanal tape.[13] The mesh arms pass through the obturator foramen while the mesh body covers the rectocele.

Transischioanal Free Implants

That technique of mesh placement was first described by Von Theobald et al.[43] A rectangular mesh covering the rectovaginal space (both centrally and laterally) is sutured at the level of the vault (or the posterior part of the uterine cervix) to a posterior transischioanal tape, as initially described by Petros,[13] passing in the space between the levator ani muscle and the sacrospinous ligament.

The transischioanal technique is currently the most widely used.[49,51-54] Other authors use a modified passage of the posterior arms of the mesh directly through the sacrospinous ligaments on both sides. Two supplemental arms can be used for a perineal tension-free fixation (Fig. 9.4).

Fixed Implants

Fixation to the Levator Ani Muscle

That technique of mesh placement was described and only used by Foulques.[59] A rectangular mesh with no arm is sutured with nonabsorbable sutures directly to the iliococcygeal part of the levator ani muscle.

Fixation to the Sacrospinous Ligament

That technique of mesh placement was first described by Dwyer et al.[34], and also used by others.[64] A midline incision from the perineum to the vaginal apex is made and the vagina detached from the rectum with sharp dissection, which is extended laterally to the ischiorectal fossa and

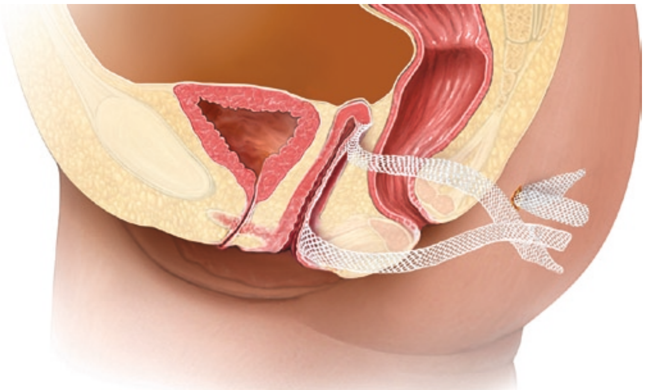

Fig. 9.4 Posterior repair with a transischioanal kit (Reproduced from CR Bard Inc. With kind permission)

superiorly onto the sacropinous ligament. If an enterocele is present, the sac is dissected out and opened and then closed by high ligation using absorbable sutures. Any fascial defect in the rectovaginal septum is repaired using absorbable suture. Mesh is fashioned in a Y shape. The arms of the Y are placed onto the sacrospinous ligaments bilaterally with the main body of mesh overlaying the repaired rectovaginal fascia and the perineal body. The fixation of the posterior arms can be done either with absorbable[34] or nonabsorbable[64] sutures. The mesh could be stabilized with absorbable sutures placed superiorly, laterally, and onto the perineal body. Rectal examination is performed routinely to exclude damage or the inadvertent placement of the sutures into the rectum.

Comparative Outcomes Between Free and Fixed Implants

In the Literature

In order to compare outcomes in regard to the technique, only studies published in the last 5 years (2003–2008) using polypropylene implants were analyzed.

Anterior Compartment

Studies using polypropylene implants vaginally for cystocele repair report generally favorable medium-term (up to 37 months of mean follow-up) anatomic outcomes, that is, anatomical correction of the cystocele (stage 0 or 1) between 64% and 100% (Table 9.1).[8-10,34-61] Only three studies have reported anatomical success rates lower than 85%, after real free[37] or retropubic free techniques.[45,46]

Three randomized controlled trials have recently shown significant better anatomical outcomes using polypropylene implant, one with real free technique[8] and two with transobturator techniques.[9,10]

Vaginal erosion (up to 21%) and several major complications directly due to the implant were reported (pelvic abscess, mesh infection, reoperation for mesh contraction, vesicovaginal fistula, and dyspareunia). However, most of these studies are heterogeneous, included patients numbers are small and follow-up is limited. Technically, there is no comparison between techniques of mesh placement. Although these studies do not permit any robust recommendation of the use of one particular surgical technique with free or fixed implant, real free or retropubic free techniques were the only ones to have anatomical success rates lower than 85%.

Posterior Compartment

Studies using polypropylene implants vaginally for rectocele repair report generally favorable medium-term (up to 29 months of mean follow-up) anatomic outcomes, that is, anatomical correction of the rectocele (stage 0 or 1) between 82% and 100% (Table 9.2).[34,38,40,43,46,49,51-54,59,62-64] However, to date, there is still no evidence demonstrating that posterior nonabsorbable implant, whatever the technique used, improves outcomes compared with traditional techniques (posterior colporraphy, site-specific repair). There are few prospective studies and no randomized trial. Furthermore, vaginal erosion (up to 19%) and several major complications directly due to the implant were reported (pelvic abscess, mesh infection, fascitis with necrosis, rectovaginal fistula, and dyspareunia).

Unfortunately, these studies are heterogeneous, series are small and follow-up is limited. Furthermore, no study has well-evaluated rectocele on defecography, and functional assessment on defecation is also very limited. Technically, there is no comparison between techniques of mesh placement.

These studies do not permit any valid conclusions but serve as interesting pilot studies on the plausibility of rectocele repair using synthetic implant. However, it is not possible to recommend one surgical technique with free or fixed implant.

French Ugytex Multicentre Study: 3-Year Comparative Results Between Free and Fixed Implants

We carried out a large multicenter prospective study to investigate the effects of a low-weight polypropylene mesh protected by an absorbable hydrophilic film in prolapse repair by vaginal route. Short-term results have already been published.[65] The 3-year analysis was performed comparing the different techniques used by surgeons in the different centers.

Methods

Between March 2003 and June 2004, 230 consecutive patients with symptomatic anterior or posterior vaginal wall prolapse were recruited in a prospective multicenter study involving 13 French gynecological or urological departments (four University, three public, and six private hospitals) and 18 surgeons experienced in vaginal prolapse repair. The preoperative evaluation included a medical history and an urogynecological examination. All departments were using a

Table 9.1 Comparative outcomes between free and fixed implants in the literature for anterior compartment

Surgical technique (free or fixed)	Authors	Year	Study design	Mesh	*n*	Mean follow-up (months ± SD or [range])	Anatomical success (Ba point <–1) (%)	Vaginal erosion *n* (%)	Other complications
Real free implants	Dwyer et al.[34]	2004	Retrospective	Atrium	47	29 [6–52]	89.4	3 (6.4)	2 de novo dyspareunia
	Bader et al.[35]	2004	Retrospective	Gynemesh	40	16.4 [12–24]	95	3 (7.5)	
	Milani R et al.[36]	2004	Prospective	Prolene	32	17	94	4 (13)	Dyspareunia increased by 20%
	Ng et al.[37]	2006	Retrospective	Prolene	37	14.4–19.2 [2–32]	75.7	0	1 abscess (2.7%) 1 hematoma (2.7%) 2 blood transfusion (5.4%)
	Sola et al.[38]	2006	Retrospective	Gynemesh Soft	20	[1–8]	100	0	
	Hiltunen et al.[8]	2007	Multicentric RCT	Parietene	104	12	93.3	18 (17.3)	1 bladder injury (1%) 1 reoperation (1%) 1 infection (1%)
	Jo et al.[39]	2007	Prospective	Gynemesh	38	23.4	94.3	0	
	Carey et al.[40]	2008	Prospective	Gynemesh Soft	95	12	90	4 (4.2)	
Retropubic free implants	Zargar et al.[41]	2004	Retrospective	Prolene	31	32	100	3 (9.4%)	Dyspareunia (12.9%)
	de Tayrac et al.[42]	2006	Retrospective	Gynemesh	63	37 [24–60]	89.1	5 (9.1)	Dyspareunia (16.7%)
	von Theobald et al.[43]	2007	Retrospective	Surgipro	73	19 [9–31]	89	2 (2.7)	4 vesical injury (5.5%) 2 retropubic hematomas (2.7%)
	Granese et al.[44]	2007	Prospective	Prolene	177	24	89	9 (5)	Dyspareunia (1%)
	Cervigni et al.[45]	2008	Prospective	Marlex	218	38	75.7	27 (12.3)	1 rectal injury (0.5%) 1 blood transfusion (0.5%) De novo dyspareunia (9.6%)
	Milani AL et al.[46]	2008	Prospective	Ti-Mesh	71	9	64	4 (5.6)	1 urethral injury (1.4%)
Transobturator free implants	Eglin et al.[48]	2003	Retrospective	Polypropylene	103	18 [2–34]	97	5 (5)	
	Fatton et al.[49]	2007	Retrospective Multicentric	Prolift	81	6	97.2	5 (4.7)	1 bladder injury (2.3%) 2/110 hematomas (1.8%)
	Sergent et al.[50]	2007	Prospective	Parietex (two arms transischioanal)	103	32 [12–53]	97	17 (16)	Dyspareunia (13%) 1 pelvic hematoma (1%) 1 rectal injury (1%) 3 blood transfusions (2.9%)
	Altman et al.[51]	2007	Prospective	Prolift	92	2	87	1 (1.1%)	3 bladder injuries (3.3%)
	Flam[52]	2007	Prospective	Prolift	55	3	94.5	0	

	Gauruder-Murmester et al.[53]	2007	Prospective	Perigee	120	12	93	4 (3)	
	Sivaslioglu et al.[9]	2008	Monocentric RCT	Parietene	45	12	91	3 (6.9)	2 de novo dyspareunia (4.6%)
	Nguyen et al.[10]	2008	Monocentric RCT	Perigee	38	12	89	2 (5)	1 blood transfusion (3%) 1 transient leg pain (3%) De novo dyspareunia (9%)
	Abdel-Fattah et al.[54]	2008	Retrospective	Prolift, Perigee	189	3	96.3	29/289 (10)	3 bladder injuries (1.6%) 2/289 infections (0.7%) Dyspareunia (4.5%)
	Shek et al.[55]	2008	Retrospective	Perigee	46	10 [2–24]	87	3 (6.5)	
	Nauth et al.[56]	2008	Retrospective	Gynemesh, Prolift, Perigee	85	[2–8]	90.4	5 (5.9)	2 blood transfusion (2.4%) 1 infected hematoma (1.2%) dyspareunia (24.3%)
Fixation to ATFP	Hung et al.[57]	2004	Prospective	Prolene (four-corner)	38	21 [12–29]	86.8	4 (10.5)	1 retropubic hematoma (2.6%) 2 blood transfusion (5.3%)
	Bai et al.[58]	2007	Prospective		28		100	1 (3)	
	Foulques[59]	2007	Retrospective	Gynemesh	317	24 [3–48]	90.9	62 (19.5)	1 vesico-vaginal fistulae (0.3%) 1 infection (0.3%) 4 reoperation for contraction (1.3%) Dyspareunia (24%)
	Handel et al.[60]	2007	Retrospective	Polypropylene	30	13.5	94	6 (21)	
Fixation to SS	Amrute et al.[61]	2007	Retrospective	Polypropylene (two retropubic arms)	76	30.7 ±1.7	94.8	2 (2.1)	Dyspareunia (9.6%)

ATFP arcus tendineus fascia pelvis, *SS* sacrospinous fixation, *RCT* randomized controlled trial

Parietene (Sofradim-Covidien, France)

IVS, Surgipro (Covidien, France)

Gynemesh, Gynemesh Soft, Prolene, Prolift (Ethicon, France)

Atrium (Hudson, New Hampshire, USA)

Perigee (AMS)

Table 9.2 Comparative outcomes between free and fixed implants in the literature for posterior compartment

Surgical technique (free or fixed)	Authors	Year	Study design	Material	*n*	Mean follow-up (months ± SD or [range])	Anatomical success (Bp point <–1) (%)	Vaginal erosion *n* (%)	Other complications
Real free implants	Milani et al.[62]	2005	Prospective	Prolene	31	17	94	2 (6.5)	1 pelvic abscess Dyspareunia increased by 63%
	Sola et al.[38]	2006	Retrospective	Gynemesh Soft	22		100	0	
	Carey et al.[40]	2008	Prospective	Gynemesh Soft	95	12	93.8	4 (4.2)	1 rectal injury (1.1%)
	Milani et al.[46]	2008	Prospective	Ti-Mesh	71	9	82	4 (5.6)	
Low transobturator free implants	Sentilhes et al.[63]	2006	Retrospective	Ugytex + Post IVS	14	13 [3–32.9]	100	0	0
Transischioanal free implants	von Theobald et al.[43]	2007	Retrospective	Surgipro + Post IVS	87	19 [9–31]	100	1 (1.1)	1 rectal injury (1.1%) 1 mesh infection (1.1%) 1 dyspareunia
	Fatton et al.[49]	2007	Retrospective Multicentric	Prolift	81	6	98.2	5 (4.7)	Dyspareunia (13%)
	Altman et al.[51]	2007	Prospective	Prolift	38	2	91	1 (2.6%)	1 rectal injury (2.6%) 1 hematoma
	Flam[52]	2007	Prospective	Prolift	55	3	100		
	Gauruder-Murmester et al.[53]	2007	Prospective	Apogee	120	12	100	4 (3)	
	Abdel-Fattah et al.[54]	2008	Retrospective	Apogee	181	3	96	29/289 (10)	2 rectal injuries (1.1%) 1/289 fasciite with necrosis (0.3%) Dyspareunia (4.5%)
Fixation to LA muscle	Foulques[59]	2007	Retrospective	Gynemesh	317	24 [3–48]	95.3	62 (19.5)	Dyspareunia (24%)
Fixation to SS	Dwyer et al.[34]	2004	Retrospective	Atrium	50	29 [6–52]	100	6 (12)	1 rectovaginal fistula 1 de novo dyspareunia
	de Tayrac et al.[64]	2006	Retrospective	Gynemesh + Prolene	26	22.7±9.2	92.3	3 (12)	De novo dyspareunia (7.7%)

LA levator ani, *SS* sacrospinous fixation
Ugytex (Sofradim-Covidien, France)
IVS, Surgipro (Covidien, France)
Gynemesh, Prolene (Ethicon, France)
Atrium (Hudson, New Hampshire, USA)

standardized case report form. Clinical evaluations were performed using the International Pelvic Organ Prolapse staging system (POP-Q), assessing anterior vaginal wall prolapse, uterine or vaginal vault prolapse, and posterior vaginal wall prolapse on maximum Valsalva effort in the seated semi-lithotomy position. In order to evaluate symptoms and quality of life, each patient was asked to answer the French version[66] of the validated Pelvic Floor Distress Inventory (PFDI) and the Pelvic Floor Impact Questionnaire (PFIQ).[67] The Urinary Dysfunction Measurement Scale (MHU)[68] and the presence and severity of dyspareunia were also investigated. Each patient recruited had at least a symptomatic vaginal wall prolapse at stage 2–4 in the International Pelvic Organ Prolapse staging system (Ba or Bp ≥ -1) and an impairment of her quality of life.

All patients were operated by the vaginal route using a specially designed prosthetic mesh (Ugytex™, Sofradim-Covidien, Trévoux, France). It is a low-weight (38 g/m²) and highly porous (89% of average porosity and pores over 1.5 mm) polypropylene monofilament mesh offering tissue ingrowth and connective differentiation. Furthermore, the mesh is coated by a hydrophilic film composed of atelocollagen, polyethylene glycol, and glycerol. The absorbable coating protects delicate pelvic viscera from the risk of acute inflammatory reaction during the healing inflammatory peak.[69]

In the operating room, the patient was prepared for surgery in the dorsal lithotomy position and with strict aseptic conditions. After a vertical midline incision or a horizontal minimal invasive incision close to the uterine cervix, the vaginal epithelium was grasped on both sides and the fibromuscular layer was sharply dissected laterally to the level of the descending pubic rami for cystocele and to the level of the ischial spine for rectocele. We did not perform any anterior nor posterior fascial plicature before the mesh placement. The prosthetic mesh was prepared with strict aseptia. Anteriorly, the mesh was implanted either with two arms into the retropubic space[42], with four arms into the obturator foramen[48], or directly sutured to the ATFP in both sides as it was described by others.[57-60] Posteriorly, the mesh was implanted either with two arms sutured to the sacrospinous ligaments[64], or mainly with two arms via the transischioanal route as it was described by others.[43,49,51-54] In all cases, the mesh was adjusted to avoid any fold and 2/0 absorbable polyglactin sutures were also used in all groups. The excess vaginal skin was not excised in order to avoid direct contact between the vaginal scar and the mesh during the postoperative scar formation. The anterior vaginal skin was closed with continuous 2/0 polyglactin sutures. A Foley catheter was introduced at the beginning of the procedure and removed after 48 h. Intraoperative antibiotic prophylaxis was systematically administrated.

Concomitant procedures such as vaginal hysterectomy or sacrospinous suspension have been performed according to each surgeon. When patient had associated preoperative stress urinary incontinence, a suburethral mesh was placed after the anterior vaginal skin closure, by a separate incision. Intra- and postoperative complications were recorded on a standardized case report form. Immediate postoperative complications were defined as those occurring during the first 30 days.

Postoperatively, patients were instructed to rest for 2 weeks, they were allowed to return to work after 4 weeks and to play a sport or to have sexual intercourse after 6 weeks. The follow-up was scheduled at 6 weeks, 6 months, and 1 year. At each visit, urogynecological examination was performed using the POP-Q system on maximum Valsalva effort in the seated semi-lithotomy position. Objective anatomical cure was defined when the anterior vaginal wall for the cystocele and the posterior vaginal wall for the rectocele were at stage 0 (optimal outcome) or 1 (satisfactory outcome). Postoperative functional results for symptoms, quality of life, and sexuality were evaluated using the PFDI and the PFIQ self-questionnaires, the MHU and questions related to presence and severity of dyspareunia.

Statistical analysis was based on the Mann–Whitney test for nonparametric continuous variables and the chi-squares test or Fisher's exact test for categorical variables. *P* value <0.05 was considered significant.

Results

Overall Cohort

Mean age was 62.9 years (ranging from 33 to 91), mean parity was 2.8 (ranging from 0 to 11), and mean BMI was 25.6 kg/m² (ranging from 14.5 to 42.6). Thirty-five patients had undergone previous surgery for prolapse (15.3%) and 65 had previous hysterectomy (28.4%). Mean operative time was 81 min (ranging from 20 to 180). Mean hospital stay was 4.5 days (ranging from 1 to 23).

The overall intraoperative complication rate was 2.6% (6/230), including two bladder injuries during the cystocele dissection, one rectal injury during the rectocele dissection, two hemorrhages, and one vaginal sulcus perforation. All these complications were immediately identified and treated without any consequences. Five major immediate postoperative complications were reported (2.2%): four noninfected and one infected hematomas. One noninfected hematoma was evacuated at day 2, leaving without removing the mesh and the infected hematoma has necessitated a partial excision of the posterior part of the mesh at day 7.

3-Year Results

The present report is based on the analysis of the 159 patients evaluated with at least 24 months follow-up (69.1% of the all cohort).

At a mean follow-up (±SD) of 37.7 ± 7 months, in the study population ($n = 159$), the overall cure rate for cystocele was 127/143 (88.8%) and the overall cure rate for rectocele was 80/84 (95.2%). Symptoms and quality of life questionnaires evaluation have shown a highly significant postoperative improvement, in comparison to preoperative results, with respective POP-DI scores at 25.3 versus 88/300 ($p < .001$) and POP-IQ scores at 15.1 versus 60.7/300 ($p < .001$).

Concerning sexuality, 77 out of 92 sexually active patients had normal postoperative sexual intercourse, 14 had mild to moderate dyspareunia (15.2%) and one patient had severe dyspareunia (1.1%).

The overall rate of vaginal erosions due to the mesh was 13.8% (22/159). Ten erosions were diagnosed after the 2-year visit. Patients with concomitant hysterectomy had more chance to develop vaginal erosion than patients without, respectively in 13/56 (23.2%) and 9/103 (8.7%) ($p = .01$). Overall, the chance for a patient to have a second procedure for erosion was 10.1% (16/22 erosions necessitating a partial excision of the mesh). There was neither postoperative fistula nor mesh infection among patients with vaginal erosion during the follow-up.

Comparative Analysis According to the Technique of Mesh Placement

In order to compare the different techniques of mesh placement, we performed a retrospective comparative analysis of outcomes according to the technique used in the different centers. For anterior repair ($n = 208$), 142 meshes were placed transobturatorly in a tension-free fashion, 32 were free into the retropubic space, 31 were fixed to the ATFP, and only three were totally free. For posterior repair ($n = 142$), 132 meshes were placed transischionally in a tension-free fashion, five were fixed to the sacrospinous ligament, and five were totally free. Therefore, because of the small sample sizes of several groups, we decided to compare only three techniques for the sole anterior compartment.

Baseline characteristics between implantation groups (retropubic free, transobturator tension-free, and fixed to ATFP) were comparable, except for previous hysterectomy (fewer in the transobturator group) and concomitant posterior repair (fewer in the fixed group) (Table 9.3). The transobturator technique took more time to be performed. Although follow-up was shorter and rate of loss to follow-up higher in the transobturator group, anatomical success was clearly poorer after the retropubic free technique, in comparison to the two other ones, with respective cure rates of 69, 90.1, and 96.6% (Table 9.4). However, reoperation rates were comparable between groups. Intra- and postoperative complications (including vaginal erosions) were comparable

Table 9.3 Comparative analysis between free and fixed implants in the Ugytex cohort for anterior compartment. Baseline characteristics and concomitant procedures

	Retropubic free implants *n* = 32	Transobturator tension-free implants *n* = 142	Fixed implants *n* = 31	*p*
Mean age	63	62.6	67	
Mean Body Mass Index (kg/m^2)	25	25.6	26.9	
Mean parity	2.5	2.8	3.6	
Previous surgery				
Previous surgery for prolapse	4 (12.5)	16 (11.3)	2 (6.5)	.69
Previous hysterectomy	14 (43.8)	29 (20.4)	11 (35.5)	.01
Preoperative cystocele				
Stage 0	0	1 (0.7)	0	
Stage 1	0	9 (6.3)	0	
Stage 2	17 (53.1)	50 (35.2)	6 (19.4)	
Stage 3	14 (43.8)	63 (44.4)	25 (80.6)	
Stage 4	1 (3.1)	19 (13.4)	0	
Concomitant procedures				
Hysterectomy	9 (28.1)	61 (42.9)	9 (29)	.13
Posterior repair	24 (75)	111 (78.2)	5 (16.1)	<.001
Mean operative time (minutes)	68.7	92	52.6	

n (%)

Table 9.4 Comparative outcomes between free and fixed implants in the Ugytex cohort for anterior compartment

	Retropubic free implants *n* = 29	Transobturator tension-free implants *n* = 86	Fixed implants *n* = 29	*p*
Mean follow-up (months)	32.9	25.8	32.9	
Lost to follow-up	3 (9.4)	56 (39.4)	2 (6.5)	
Anatomical success	20 (69)	78 (90.1)	28 (96.6)	.004
Reoperation for recurrence in all compartments	0	7 (8.1)	2 (6.9)	
Intraoperative complications				
Intraoperative bladder injury	1	3	0	
Vaginal perforation	1 (TOT)	0	0	
Hemorrhage	1 (hysterectomy)	1	0	
Postoperative pain				
Postoperative pain at day 3	11 (37.9)	24 (27.9)	0	
Postoperative pain at 1 months	2 (6.9)	19 (22.1)	3 (10.3)	
Postoperative pain at 6 months	0	12 (14)	0	
Postoperative complications				
Postoperative hemorrhage/hematoma	0	4	0	
Blood transfusion	0	1	0	
Infection on skin incisions	0	1	0	
Vaginal erosion	7 (24.1)	18 (20.9)	6 (20.7)	.13

n (%)

between groups, even if we observed slightly more postoperative hemorrhage in the transoburator group. Postoperative pain was not more severe in the fixed group.

Although several bias exist in that comparison (retrospective analysis, different operators for different techniques, different sample sizes), this is the first available comparison of three techniques for anterior repair vaginally using a mesh placement. Randomized comparison should be of great interest for urogynecologists.

Conclusion

In the last 10 years, many techniques of synthetic mesh placement were proposed in vaginal reconstructive surgery, for both anterior and posterior repair. The most widely used techniques are currently the transobturator mesh with four arms in the anterior compartment and the transischioanal mesh with two arms in the posterior compartment. These techniques follow the concept of tension-free, consisting of the passage of the mesh into tissues without dissection. The mesh fixation is obtained by surround tissue maintenance 25 days after surgery. Although the published studies do not permit any robust recommendation of the use of one particular surgical technique with free or fixed implant, poorer results were only observed after real free or retropubic free techniques.

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A Comparative Analysis of Biomaterials Currently Used in Pelvic Reconstructive Surgery

10

Richard I. Reid

Biomaterials in Overview

The use of prosthetic materials and trocar driven kits in prolapse repair has risen sharply, despite a paucity of safety and efficacy data. Much of this impetus has been driven by marketing claims, not prudent practice. There is an abundance of short term clinical series which over-emphasize anatomic outcome but under-emphasize the morbidity potential of these devices. Biomaterials are regularly being implanted by practitioners who have only a rudimentary understanding of the principles of prosthetic repair. If the same degree of unpreparedness was tolerated in the Aviation industry, one wonders what would become of air travel. There is an urgent need for a return to critical analysis and common sense. While the concept of evidence based medicine is laudable, it is just as important to recognize that surgery is a craft. The effect of implanting a given biomaterial is largely determined by its biochemical properties. Before looking for statistical guidance, gynecologists must first ensure that their mesh usage does not violate these basic biochemical rules and that their decision making remains in accord with established surgical principles. This chapter revisits wound biochemistry in a simple way – to explain why some biomaterials (by their very nature) produce good results, while other biomaterials (again by their very nature) produce unreliable or even bad results. Pertinent animal studies and new concepts from regenerative medicine are used to formulate some concrete surgical corollaries for mesh usage. Finally, tissue engineering principles that will likely reshape reparative surgery in the coming decade are examined.

A Brief History of Biomaterials

Over the years, surgeons from all disciplines have searched for ways to replace missing tissues or repair damaged ones. Current choices lie between thermoplastic polymers, organic polymers, and biosynthetic constructs:

Bioreactive materials

Until the mid-twentieth century, surgeons had only bioreactive materials like *catgut* (denatured collagen filaments from sheep or cow intestine) or *silk* (a thread prepared from the dried sap of mulberry trees, harvested from silkworm cocoons). These fibers performed modestly well as single strand sutures, but their use en masse to buttress tissue defects was out of the question.

Thermoplastic polymers

The discovery in the 1930s that certain short-chain hydrocarbon monomers could be elaborated into strong polymers spawned the "plastics" industry (i.e., the manufacture of thermoplastic polymers that could be molded into different shapes when heated). Four synthetic polymers are commonly used in soft tissue surgery:

- *Nylon:* Nylon is a linear polyamide synthesized by joining together thousands of small hydrocarbons with carbon-oxygen-nitrogen (peptide) covalent bonds, through a condensation reaction that removes water molecules (Fig. 10.1a). Because of its multiple peptide bonds, nylon bears a stereochemical resemblance to such protein fibers as silk and wool. When first adapted for use as suture material just before World War II, nylon was something of a revelation. Nylon fibers were stronger, more durable, and much less reactive than either catgut or silk. Its successful use as a darn began the modern era of prosthetic hernia repair.[1] However, nylon's linear polyamide structure makes it susceptible to degradation by amide hydrolysis (the reverse of the condensation reaction used to synthesize the polymer), making it unsuitable for elaboration into an alloplastic mesh.
- *Polypropylene*: Polypropylene is made by polymerizing propylene (a gaseous byproduct of petroleum refining) in

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the presence of a catalyst, under carefully controlled heat and pressure. Thousands of propylene molecules are added sequentially, until the chain reaction is terminated. Chemical and mechanical properties vary according to the molecular weight (i.e., length) of the final polypropylene chain. The polymerization reaction involves only carbon and hydrogen atoms, joined by multiple unsaturated bonds; hence, polypropylene is completely nonwettable and very stable (Fig. 10.1b). When used as single strand monofilament sutures, polypropylene is sufficiently biocompatible that any surrounding inflammatory reaction seldom reaches clinical significance. Unfortunately, aggregation of polypropylene fibers into a mesh exceeds the critical mass that a tissue will tolerate, without triggering a foreign-body reaction. Being hydrophobic, the resulting scars tend to be quite rigid – but infection resistant.

- *Polyesters*: Polyester means a polymer containing an ester functional group in its main chain. There are many polyesters; however, the term "polyester" is often used to refer to a specific material, made by the polymerization of phthalic acid with ethylene glycol, to form *polyethylene terephthalate* ("PET") (Fig. 10.1c). PET has been the polyester most

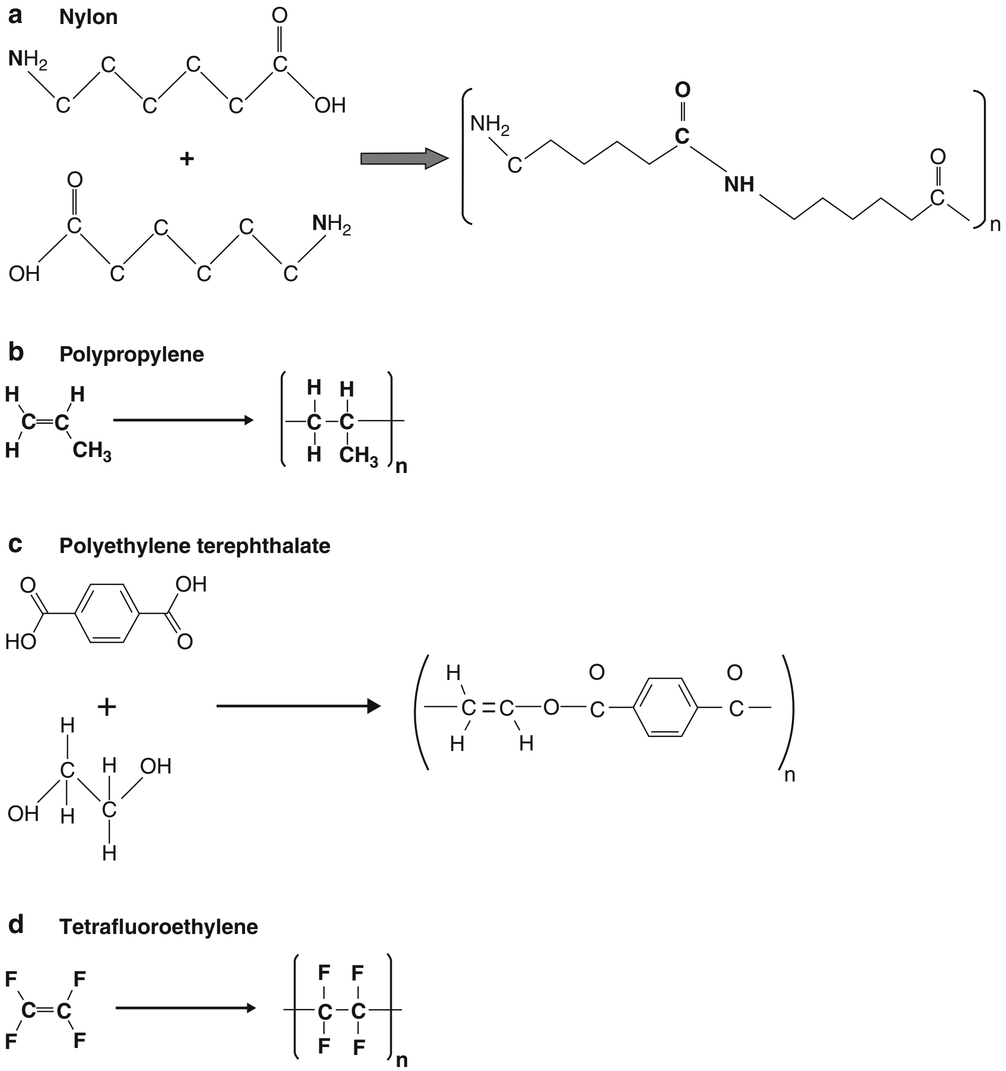

Fig. 10.1 Chemical formulae. (**a**) A diagram showing how the condensation reaction removes a water molecule from a pair of six-carbon amides (oriented head to tail in this diagram), thereby creating a peptide bond. Repeating this reaction many times will polymerize these monomers into a nylon molecule. Nylon can be degraded by hydrolysis, which essentially reverses the polymerization reaction. (**b**) A diagram showing the polymerization of propylene gas into a polypropylene fiber. Note how the carbon and hydrogen atoms are tightly packed, with no ready spaces for adherence of water molecules. Hence, polypropylene is quite hydrophobic. (**c**) A diagram showing the polymerization of terephthalic acid with ethylene glycol to form polyethylene terephthate (*PET*). Note how the component atoms are much more dispersed, leaving spaces for adherence of water molecules by hydrogen bonding. (**d**) A diagram showing the polymerization of tetrafluoroethylene to form (poly)tetrafluoroethylene (*PTFE*). The high electronegativity of fluorine makes PTFE chemically inert and extremely nonadherent. In its original chemical form, PTFE can be spun into a multifilamentous mesh (Teflon® and Dacron®). The expanded form of PTFE was originally produced as an Amid type II microporous mesh (Gore-Tex®), but is now more commonly employed as an Amid type IV submicronic antiadhesion membrane in composite hernia and cardiac implants

commonly used in surgery, either coated and spun into braided sutures (Ethibond®, Somerville, NJ; Tycron®, Tyco Healthcare, Greenwich, NJ) or woven into multifilament mesh (Mersilene®, Ethicon, Somerville, NJ; Dacron®, DuPont, Kingston, NC). Another polyester commonly used in soft tissue surgery is *polytetrafluoroethylene* (Teflon®, DuPont, Kingston, NC). PTFE is made by polymerizing carbon and fluorine with multiple strong carbon–fluorine bonds (Fig. 10.1d). It is very inert and friction reducing, but relatively susceptible to foreign-body inflammatory reactions.

As a group, polyesters are delicate hydrophilic fibers with excellent mechanical and anatomic conformation properties – which is why they have been so successful in clothing manufacture. Polyesters have nice handling properties and tend to form relatively soft scars. However, there is a downside. Being wettable makes polyesters much more susceptible to bacterial adherence than hydrophobic molecules. This weakness is compounded by the fact that bacteria (1 μm) easily enter the interstices in woven multifilament mesh. Macrophages and natural killer cells (~21 μm) are too large to penetrate such small spaces.[2] Colonizing bacteria can therefore persist in sanctuary sites, potentially releasing lytic (erosive) toxins or flaring up as a delayed mesh infection (see Chap. 3, *Principles for Synthetic Mesh Hernia Repair*). Recently, a new generation of more sophisticated knit-weaves of multifilament polyester have been engineered (Parietex®, Tyco Healthcare, Greenwich, NJ), to again provide hernia surgeons with soft but infection-resistant hydrophilic implants. These new products have interstices >10 μm, and are often protected by collagen or polyglactin films.

- *Expanded polytetrafluoroethylene:* Expanded polytetrafluoroethylene (Gore-Tex®, W. L. Gore & Associates, Inc, Newark, DE) is a microporous polytetrafluoroethylene mesh, with a microstructure characterized by tiny nodes and interconnecting fibrils. Gore-Tex sutures and mesh (expanded-PTFE) have entirely different surgical properties to the parent chemical compound, PTFE. Gore-Tex sutures have found favor with cardiovascular surgeons, but their use in sacropexy was associated with an ×8.6 increase in odds ratio of suture erosion.[3] Likewise, use of Gore-Tex mesh in pioneering prolapse or incontinence studies was associated with a 30% rejection rate, compared to 19% for Dacron®.[4] In comparison, typical erosion rates for medium-weight mesh being used at this time in France ranged between 6% and 12%.[5] These results are not surprising, given that Gore-Tex mesh is an Amid type II compound with <10 μm pores. As such, Gore-Tex® mesh does not lend itself for use in tension-free prolapse repair. However, when formulated as a submicronic mesh, Gore-Tex® does have a useful role as an Amid type IV an inert pericardial barrier in cardiac surgery or an adhesion prevention barrier for ventral hernia repair.

Acellular organic polymers

This category consists of two distinct generations of organic polymers, each of which has unique surgical properties.

- *First-generation allografts and xenografts*: In the 1980s, biomaterials scientists decellularized a variety of allografts and xenografts,k and then cross-linked the component collagen fibers by metallic salt precipitation (a process analogous to tanning leather). Collagen was further denatured by sterilization with gamma or electron beam irradiation. The rationale behind leatherizing these collagen grafts with harsh chemical precipitants was to retard graft resorption and to suppress any potential host immune reaction (by hiding residual antigens). In reality, this strategy converted a potentially interactive graft into a denatured scaffold. Such adulterated implants are treated as "dead tissue" by the host immune response, provoking a destructive inflammatory reaction. All in all, the initial hopes of transforming these new biomaterials into "permanent" organic implants were never realized.
- *Second-generation allografts and xenografts*: To circumvent these difficulties, scientists of the early nineties prepared some collagen scaffolds in their natural state. Unwanted tissue layers were mechanically teased away from the target connective tissue, and residual animal DNA (within fibroblasts and endothelial cells) was extracted by gentle osmotic or enzymatic leaching. This manufacturing method delivered a complex mixture of proteins, glycoproteins,[*] glycosaminoglycans, proteoglycans,[†] and growth factors, still arranged in a unique, tissue-specific architecture. Such implants differ from a two-dimensional synthetic mesh in their preservation of a three-dimensional architecture, and from a "leatherized biomesh" in their preservation of a viable noninflammatory matrix.[6] These degradable scaffolds are mechanically strong enough to support the wound during early healing and bioactive enough to repair the tissue defect by "constructive remodeling."[7,8] This is a distinctly different phenomenon from the scar formation that invariably follows implantation of a synthetic or denatured biological mesh. In constructive remodeling, the spaces vacated by the washed out animal cells are repopulated by specialized host cells and accompanying blood vessels. New host

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[*] Both glycoproteins and proteoglycans are protein–carbohydrate conjugates; however, these two classes differ markedly in chemical structure and biological actions.

Glycoproteins are lightly glycosylated proteins with relatively simple oligosaccharide side-chains. Glycoproteins are important transmembrane proteins, and they play a vital role in immune recognition and cell–cell interactions.

[†] Proteoglycans are complex, more heavily glycosylated constructs. They are made up of multiple GAG molecules, bound at right angles to a central protein core. Stereochemically, they have a brush-like 3-D shape. Although proteoglycans do contribute to tissue turgor, their main role is as regulatory molecules in cell growth, migration, and differentiation.

collagen fibers are then laid down in a mirror image of the xenogeneic collagen fibers in the absorbable scaffold. Over the succeeding months, the transplanted glycoproteins, proteoglycans, and growth factors orchestrate differentiation of this new collagen scaffold into a permanent layer of new host tissue, appropriate to the implantation site.

Biosynthetic constructs

Medical textile manufacturers have made some quite ingenious innovations to mesh design, largely to meet the needs of laparoscopic hernia repair. There are also clinical situations in prolapse repair that benefit from use of a biosynthetic construct. For example, a first-generation organic polymer has been used to isolate underlying polypropylene mesh from host inflammatory response (Avaulta®, CR Bard Inc, Murray Hill, NJ). In the future, hybrid electrospun scaffolds will likely combine the attractive mechanical features of a nanofiber synthetic with the bioactivity of naturally derived extracellular matrix components.[9]

Host Response to Implantation of a Biomaterial

What really happens when a surgeon implants a biomaterial? Basically, there are two (and only two) possible host responses, depending on the biochemical make up and structural organization of the implant. The response to all conventional biomaterials thereafter follows a predictable sequence of events, with initial foreign-body giant-cell inflammation and late fibrosis at the host–implant interface. However, specially prepared materials can alter this default response to one of tissue induction, with a downstream effect of constructive remodeling.

Synthetic mesh

Surgeons of the 1970s initially preferred uncoated polyester implants (Mersilene® or Dacron®) because of their superior handling properties and softer scar formation.[10,11] Unfortunately, their microporous and/or multifilamentous construction restricted macrophage and natural killer cell activity. Herniologists soon switched to macroporous monofilament mesh, as a more sepsis-resistant device.[12,13] Infection aside, both polyester and polypropylene evoke an initial foreign-body inflammation, which eventually moderates (but never really abates).[14-16] Mechanically speaking, healing response is shaped by two main factors: pore size/accessibility and device motion at the implantation site.[17,18] Recognition of these influences led to the Amid classification of synthetic mesh (Table 10.1 and Fig. 10.2).

Table 10.1 Synthetic surgical mesh implants

Class	Fiber type	Pore size	Polymer chemistry	Trade names	Weight (gm/m^2)
Absorbable	Multifilament	Macro	Polyglactic acid	Vicryl® mesh (Ethicon)	35
			Polyglycolic acid	Dexon® mesh (Covedin)	
Semiabsorbable	Multifilament	Macro	Polyglactic acid and polypropylene	Vypro II® mesh (Ethicon)	63
Amid Type I	Monofilament	Macro (>75 μm)	Polypropylene	Marlex® (Bard)	152
				Surgipro SPMM® (Covedin)	97
				Prolene mesh® (Ethicon)	85
				Prolite® (Atrium)	52
				Gynemesh® (Ethicon)	50
Amid Type II	Multifilament	Microporous(<10 μm)	Expanded polytetrafluoroethylene	Gore-Tex® (Gore)	N/A
Amid Type III	Multifilament	Micro/Macro	Polyethylene terephthalate	Mersilene® (Ethicon) Dacron® (Dupont)	43
			Polytetrafluoroethylene	Teflon® (Gore)	317
Amid Type IV	Imperforate sheet	Submicronic	Polydimethylsiloxane (a silicone-based elastomer)	Silastic® (Dow Corning)	N/A
			Polypropylene sheeting	Cellgard® (Hoechst Celanese)	
			Expanded polytetrafluoroethylene, with a submicronic sheet on one side +/– macroporous mesh on the other)	Preclude pericardial membrane® (Gore) Dualmesh® (Gore)	

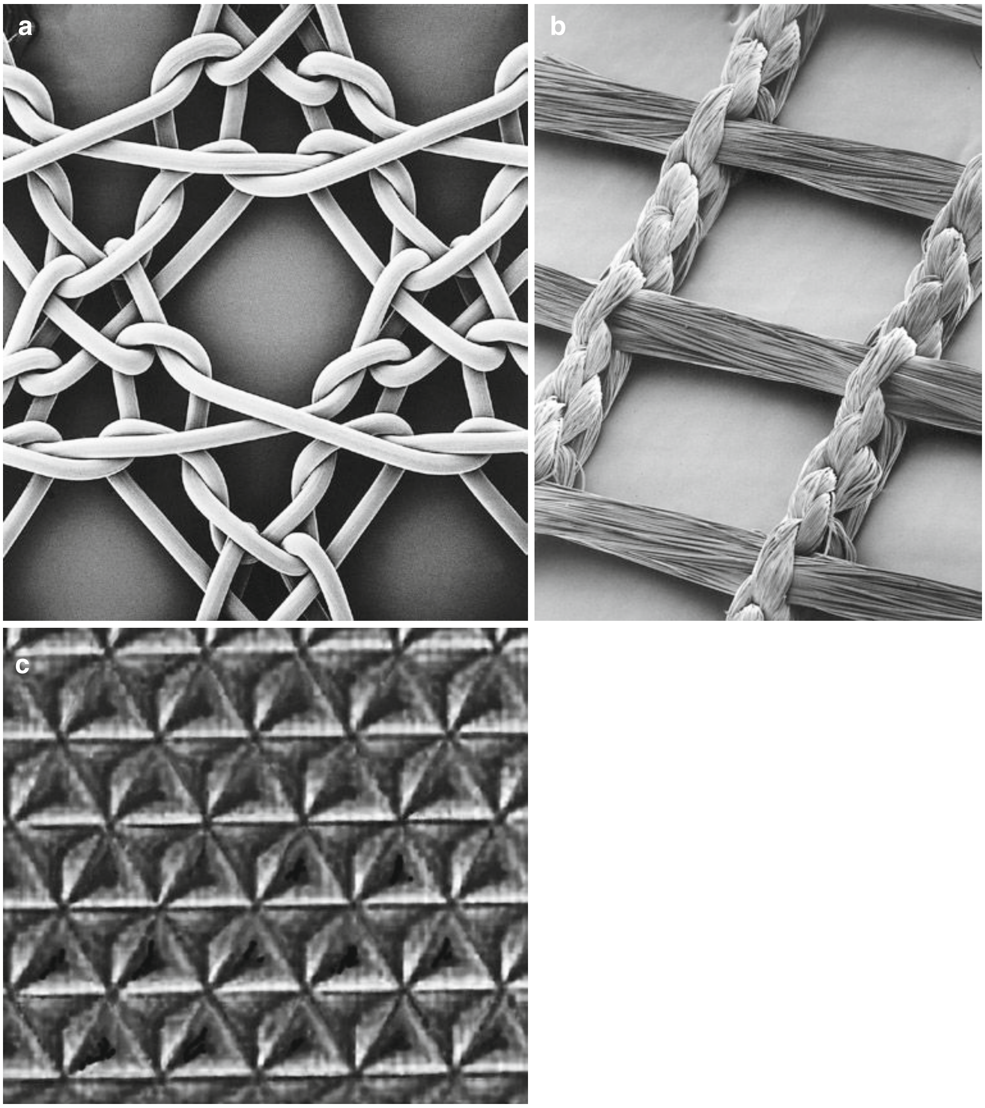

Fig. 10.2 Mesh designs. (**a**) Scanning electron microscopy of a knitted heavy weight monofilament polypropylene Amid type I mesh. (**b**) Scanning electron microscopy of a knitted multifilament PET mesh. (**c**) Scanning electron microscopy of an Amid type IV submicronic sheet

- *Amid type I mesh*: Macroporous monofilament mesh is readily penetrated by vascular and fibroblast ingrowth; scar maturation later strangles these areas of neovascularization. Thus, placing an Amid type I mesh within immobile tissues generally creates a felt-like collagenous band that is strongly attached to adjacent host tissues[19-22] (Fig. 10.3a and b). However, excessive shearing against host tissue due to poor implant fixation or repeated tissue motion can create a microbursa.[17] Despite their robust properties for prosthetic hernioplasty, Amid type I meshes have some distinct disadvantages for prolapse repair, where scarring around permanent materials is inherently morbid. This is especially true of polypropylene because its nonwettable nature and torsional rigidity lead to abrasive scars and poor wrinkle recovery.[24] Nonetheless, with good surgical judgment, net outcome can be made more beneficial than adverse. The key consideration is to limit the degree of compliance mismatch at the implantation site (see Chap. 3, *The Era of Anatomic Discovery*). An abrasive scar is generally well tolerated in the static tissues of the urogenital diaphragm[14] or sacral hollow[3,25] because large shearing forces are not generated at the implant interface. However, compliance mismatch is more problematic in the mobile tissues of the anterior or posterior compartments. Constant shearing of the vaginal walls across an abrasive mesh can have a “cheese grater” effect – creating erosions, severe cicatrization, and a risk of fistula formation. The chronic foreign-body inflammation can also cause chronic pain and dyspareunia. At this point in time, synthetic mesh complications are potentially reducible (but not fully resolvable) by improved surgical technique.[4,5,14]
- *Amid type II and III mesh*: Fibroblast and vascular ingrowth is restricted by the microporous and/or multifilamentous construction; hence, Amid types II (eg, Gore-Tex mesh) and III (eg, Mersilene and Dacron) meshes tend to encap-

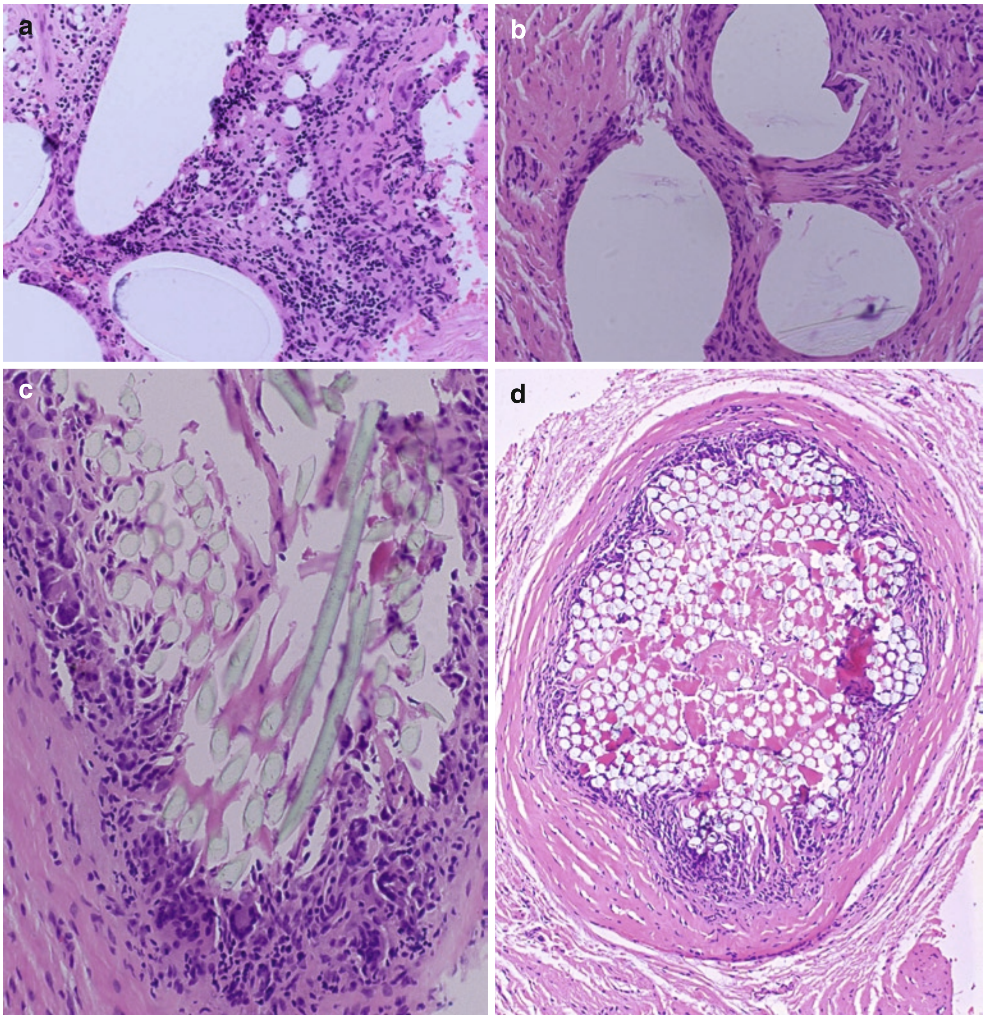

Fig. 10.3 Histology. (**a**) A low-power view of a recently implanted macroporous mesh. To the left, four strands of polypropylene are walled off within cystic spaces, surrounded by an intense inflammatory cell infiltrate of neutrophils, plasma cells, and multinucleated foreign-body giant cells. Some neovascularization can be seen to the right (H&E ×10). (**b**) Blood vessels proliferate through the pores of the macroporous polypropylene mesh, leading to "incorporation" of the implant. Intensity of the white cell infiltrate fades with time, but the foreign body inflammation never resolves, as evidenced by the three cystic foreign bodies shown in this late biopsy. Areas of neovascularization are subsequently constricted by accompanying fibroblastic ingrowth, creating a field of dense nonlaminar collagen. The absence of a GAG component is readily appreciated (H&E ×10). (**c**) An intense inflammatory cell infiltrate surrounding a recently implanted multifilament polyester mesh. Lymphocytes predominate, but plasma cells, and multinucleated foreign body giant cells are prominent (H&E ×20). (**d**) A late biopsy shows the PET strands within encapsulated by dense fibroplasia, with residual inflammation. Clinically, this PET mesh was creating substantial pain and vaginal deformity, but had not become infected (H&E ×10). (**e**) Suburethral Pelvicol™ porcine dermal graft 19 weeks after implantation. There is a mild lymphocytic reaction at the graft–host interface, together with a low level of fibroblast proliferation and neovascularization. A minimal amount of new host-derived collagen has been deposited peripherally. Centrally, the implanted acellular collagen scaffold remains essentially unaltered, as evidenced by the regular interweaving pattern of porcine collagen without any host cell infiltration. H&E ×10 magnification. (Reproduced from Gandhi et al. [23]. With permission) (**f**) Suburethral Pelvicol™ porcine dermal graft 42 weeks after implantation. The graft has been heavily infiltrated by histiocytes and multinucleated giant cells, with substantial autolysis of the implanted collagen scaffold. Repair failure appears imminent. H&E ×10 magnification. (Reproduced from Gandhi et al. [23]. With permission) (**g**) A biopsy of Surgisis® shortly after implantation (10 wks). The implanted scaffold can still be identified as dense strands of acellular collagenous tissue, which is beginning to repopulate with connective tissue fibroblasts. Because host immune response recognizes this implant as "natural", there is only a minimal degree of host inflammatory reaction (H&E ×20). (**h**) Eight months after implantation, the implanted Surgisis® has been completely remodeled into an aponeurosis-like sheet of new host connective tissue, rather than scar. Laminar collagen bundles are oriented into the parallel arrays that characterize strong physiological connective tissue. Note the spaces between these collagen bundles, which are filled with the GAG layer necessary for cell migration and effective homeostasis. No inflammation or residual porcine elements can be identified (10×). (**i**) The same biopsy, sectioned at right angles to figure 2(h). There is physiological blood vessel growth between the laminated collagen bundles, rather than random inflammatory neovascularization (10×)

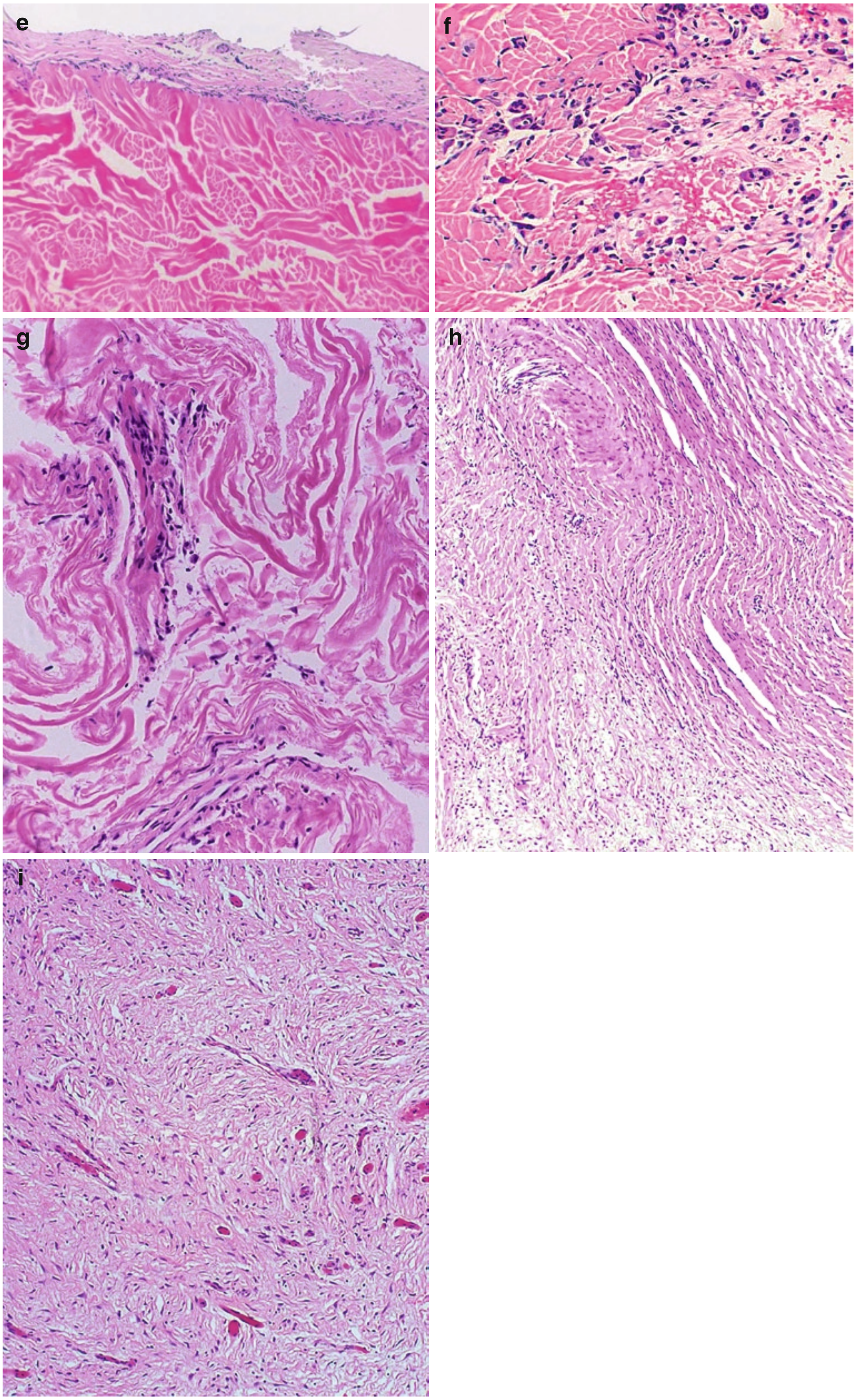

Fig. 10.3 (continued)

sulate within a minibursa, creating potentially weak anchorage sites (Fig. 10.3c and d). A second major disadvantage is that of bacterial colonization or overt mesh infection, leading to complication rates of 20–30%[4,26,27] (see Chap. 3, *The Herniology Era*).

- *Amid type IV mesh*: These materials have <1 μm pores that completely prevent cellular ingrowth. As such, they play a vital adhesion-suppressant role on the peritoneal surface of composite hernia meshes. However, Amid type IV submicronic membranes have no useful role in tension-free prosthetic repair of hernia or prolapse.

First-generation organic polymer

With the wisdom of hindsight, altering protein architecture within a xenograft is actually counter-intuitive, and created three insurmountable disadvantages. First, persistence of denatured animal collagen at the surgical site sets in train a pro-inflammatory macrophage response, engulfing the implant in a chronic foreign-body giant-cell reaction. This rules out any possibility of constructive remodeling because downstream healing events are now committed to a pathway of cicatrization.[28,29] Second, cross-linking the protein scaffold confines the host immune response to the implant surface; chemically precipitated organic polymers therefore behave like an Amid type II mesh and encapsulate (see Section Matrix Cells). Final healing is characterized by dense but poorly organized fibrous tissue, which is often weakly bonded to the body wall.[22] Third, should the inflammatory enzymes within the surrounding miniseroma succeed in degrading the cross-linked collagen scaffold, one would expect repair failure[23,28,30,31] (Fig. 10.3e and f). Such theoretic predictions have been confirmed by clinical experience. Stiff and painful[32] or eroded wounds[33-35] have been reported, attributable to the foreign-body reaction against residual denatured collagen. There are also multiple reports of poor outcomes when Tutoplast® (Coloplast Corp, Minneapolis, MN) or Pelvicol® (CR Bard Inc, Murray Hill, NJ) were used for prolapse repair.[36-38] Reoperation often showed no residual graft material at the surgical site.[39]

Second-generation organic polymer

Preservation of an architecturally normal collagen structure and still viable matrix molecules creates a "biodegradable scaffold with an ingrained information highway."[7,8,40] Research has identified "biodegradability" as the key factor in ensuring a constructive remodeling (rather than a scarring) response.[41] In a study involving C^{14}-labeled porcine small intestinal submucosa (SIS–ECM), 60% of the implant was resorbed in 30 days, and the xenogeneic collagen scaffold had been completely replaced within 90 days.[42] The absence of inflammatory or necrotic debris allows healing to be driven by an immuno-modulatory macrophage response,[28,30] creating a neomatrix that can become stronger than the preexisting native tissue[42-44] (Fig. 10.3g–i). Such repairs are also self-renewable (permanent) because the newly formed tissue is maintained by the physiological forces that drive collagen homeostasis.[45,46]

Presently available biological grafts are described in Table 10.2. Understanding the differences between these two classes of biological implants is of immense practical importance, as highlighted by a recent study comparing five biologic scaffold materials currently marketed for human rotator cuff reconstruction.[28] The two products of principal interest were made from porcine small intestinal submucosal (SIS) xenografts, one prepared by carbodiimide cross-linking and the other maintained in its native state; an autologous tissue graft was used as a control implant. All products showed an initial nonspecific mononuclear macrophage infiltrate. Thereafter, the host response differed profoundly, according to whether downstream healing events were modulated by proinflammatory (M1) or immunomodulatory and tissue remodeling (M2) macrophages (see Section Matrix Cells). By 4 weeks, the carbodiimide crosslinked form of porcine-derived SIS had induced intense foreign-body giant-cell formation. By 16 weeks, healing had progressed to the point where foci of denatured porcine collagen were encapsulated within poorly organized fibrosis. Conversely, the structurally intact SIS was almost resorbed at 4 weeks, and had remodeled into well-organized collagen. The autologous tissue graft attracted a dual M1/M2 macrophage population, leading to partial remodeling within a field of reasonably good-quality scar formation. However, it should also be noted that the tissue of origin and the manufacturing processes profoundly influence surgical properties,[30] so one cannot exactly equate different products within a particular class.

The Biochemical Make-up of Connective Tissue

Normal connective tissue consists of 10% matrix cells and 90% intercellular space. The "ground substance" is not just inert filler, as was traditionally believed. Rather, the extracellular matrix – or the "ECM," as it is called – is an active metabolic unit that maintains the crucial balance between cells and matrix. An important paradox is that the cells secrete and organize the matrix in the first instance, but the matrix thereafter governs cell behavior. This "dynamic reciprocity" between the proteins of the ECM scaffold and the cytoskeletons of the resident cells is the feedback system that controls cell growth, proliferation, differentiation, migration, and spatial orientation.

Table 10.2 Allografts and xenografts presently available for hernia and pelvic floor surgery

Brand name	Distributor	Base material	Sterilization method	Protein scaffold	Elastin content	Matrix molecules	Host response
Collagen bulking agents							
Contigen® Bard® Collagen Implant	C.R. Bard, Inc	Bovine dermis	Low-dose gamma radiation	Glutaraldehyde cross-linked and cut into small particles	Nil	Nil	Encapsulation and fibroplasia
First-generation biologicals							
Axis Tutoplast® Processed Dermis	Coloplast Corp. (Formerly Mentor Corporation)	Cadaveric human dermis	Low-dose gamma radiation	Collagen scaffold denatured by freeze drying and acetone extraction	High	Nil or inactive	Encapsulation and miniseroma formation
Suspend Tutoplast® Fascia Lata	Coloplast Corp. (Formerly Mentor Corporation)	Cadaveric human fascia lata	Low-dose gamma radiation	Collagen scaffold denatured by freeze drying and acetone extraction	Low	Nil or inactive	Encapsulation and miniseroma formation
Peri-Guard®	Synovis Life Technologies	Bovine pericardium	Polypropylene oxide and ethanol	Cross-linked with glutaraldehyde	Low	Nil or inactive	Encapsulation and miniseroma formation
FortaGen®	Organogenesis Inc	Porcine small intestine submucosa	Irradiation	Heavily cross-linked with carbodiimide	Low	Nil or inactive	Encapsulation and miniseroma formation
FortaPerm®	Organogenesis Inc	Porcine small intestine submucosa	Irradiation	Lightly cross-linked with carbodiimide	Low	Nil or inactive	Encapsulation and miniseroma formation
Pelvicol®	C.R. Bard	Porcine dermis	Irradiation	Cross-linked with hexamethylene diisocynate	High	Nil or inactive	Encapsulation and miniseroma formation
Pelvisoft®	C.R. Bard	Porcine dermis	Irradiation	Cross-linked with HMD and then fenestrated	High	Nil or inactive	Encapsulation with partial fibrovascular incorporation
Second-generation biologicals							
Bard Dermal Allograft®	C.R. Bard, Inc	Cadaveric human dermis	Low-dose gamma radiation	Natural	High	Nil or inactive	Encapsulation and miniseroma formation
FasLata® Allograft Tissue	C.R. Bard, Inc	Cadaveric human fascia lata	Low-dose gamma radiation	Natural	Low	Nil or inactive	Encapsulation and miniseroma formation
Repliform® (for urogyn) or Alloderm® (for gen surg)	Repliform® made by Lifecell but sold by Boston Scientific; Alloderm® made and sold by Lifecell	Cadaveric human dermis	None (provided nonsterile)	Natural	High	Assumed, but not documented	Constructive remodeling (proven)

(continued)

Table 10.2 (continued)

Brand name	Distributor	Base material	Sterilization method	Protein scaffold	Elastin content	Matrix molecules	Host response
Surgisis® ES	Cook Medical	Porcine small intestine submucosa	Ethylene oxide	Natural	Low	Adhesive proteins, mucopolysaccarhides and growth factors	Constructive remodeling (proven)
Xenform™ (urogyn) or SurgiMend™, (gen surg)	TEI Biosciences, sold through Boston Scientific	Fetal bovine dermis	Ethylene oxide	Natural	Low	Assumed, but not documented	Constructive remodeling (assumed)
Veritas® Collagen Matrix, now marketed as Hydrix™ XM	Developed by Synovis Surgical, but now sold by Caldera Medical	Bovine pericardium	Irradiation	Natural	Low	Assumed, but not documented	Constructive remodeling (assumed)
DermMatrix® (formerly InterXene)	American Medical Systems	Porcine dermis	Irradiation	Natural	High	Assumed, but not documented	Constructive remodeling (assumed)

American Medical Systems, West Minnetonka, MN; Boston Scientific, Quincy, MA; CR Bard, Murray Hill, NJ; Caldera Medical, Agoura Hills, CA ; Coloplast Corp, Minneapolis, MN; Cook Medical, Bloomington, IN; Lifecell Corporation, Branchburg, NJ; Mentor, Santa Barbara, CA; Organogenesis Inc, Canton, MA; Synovis Life Technologies, St Paul, MN; TEI Biosciences, Boston, MA; Tutogen Medical Inc, Alachua, FL

Matrix Cells

There are three cell series of importance in the extracellular matrix.

Mast Cells are part of the innate immune system, found in the connective tissue and in the mucous membranes. When activated, mast cells rapidly release granules that are rich in histamine, heparin, and cytokines, creating the edema and chemotactic response of early wounding. Mast cells also regulate the vascular proliferation stage of granulation tissue development.

Macrophages are white blood cells residing within the tissues; they are produced by the division of invading monocytes. Macrophages participate in both innate immunity (as nonspecific phagocytes) and adaptive immunity (by stimulating a pathogen-specific cell-mediated response). Macrophages are particularly important to ECM function, where they regulate injury response and wound healing. A phenotypic and functional polarization of the mononuclear phagocyte cell population has recently been described[47]; this is similar to the Th1/Th2 polarization‡ scheme for lymphocytes.[48] Proinflammatory, cytotoxic macrophages (signified as M1) promote pathogen killing and phagocytosis of foreign materials, thus evoking chronic inflammation and eventual scar formation. A second phenotype of macrophages (signified as M2) promotes an immuno-regulatory response, culminating in tissue repair and constructive tissue remodeling. Although morphologically indistinguishable at histology, these macrophage families can be recognized by their cell surface markers and by their cytokine and gene expression profiles. Whether an implant initially elicits an inflammatory or a tissue modulatory macrophage response has a greater impact on wound quality than the surgeon's skill with a needle and thread.

Fibroblasts are the very essence of healing. They secrete both the fibrous and adhesive proteins, and are also involved in mucopolysaccharide production.

Macromolecules

The second component of the extracellular matrix is the macromolecules, of which there are two important families in the ECM: proteins and polysaccharides.

- The protein macromolecules consist of the fibrous proteins (various collagens and keratins) and the equally important adhesive proteins (fibronectin, laminin and integrin).
- Carbohydrate-containing macromolecules can serve either a structural or a nutritional role. Structural polysaccharides exist in two forms: pure polysaccharide molecules called glycosoaminoglycans (or GAGs) and a group of GAG-protein constructs called proteoglycans (discussed in Section Sufficient Tissue Hydration to Produce Lubrication, Tissue Turgor, and Cell Migration Lanes).

Fibrous protein and structural polysaccharide molecules both play indispensable – but opposite – mechanical roles.

Proteins

Proteins comprise an ordered array of amino acids, polymerized by covalent peptide bonds. Forces acting on these peptide bonds secondarily conform the molecule into either globular or fibrous proteins. The former have a largely functional role (enzymes, hormone receptors); the latter are structural proteins (collagen and elastin). Overall, protein biology is dictated by three striking biochemical characteristics:

- The large number of covalent bonds in a protein molecule provides exceptional tensile strength (important in collagen and keratin) and good resilience (important in elastin).
- The multiple covalent bonds are powerful enough to compact proteins into remarkably strong molecules.
- Protein molecules are electron neutral, thus conferring the structural flexibility to fold into strategic tertiary and quaternary shapes – forming cell receptor sites (on globular proteins) or attachment sites (on structural proteins).

Structural proteins fulfill many key functions within the ECM: they create a strong framework that supports the overall matrix, provide attachment sites for other macromolecules, and orient vital signaling factors into the correct spatial arrangement. Any alteration in protein ultrastructure will obviously detract from ultimate wound quality. However, proteins have one prominent liability – their highly compacted structure deprives proteins of any real ability to withstand compressive forces. If tissue were made solely of protein, it would be easily crushed.

Collagens are structural proteins that have one or more domains with a right-handed triple helix conformation. In serving as the major structural protein of mammalian ECMs, the collagen family has had to evolve into quite diverse molecular patterns – such as a rope-like organization in tendons and ligaments and an interwoven mesh-like architecture in dermis and lamina propria (Table 10.3). Actually, the polypeptide structure of the basic monomeric collagen molecule (tropocollagen) is constant throughout the entire family. The tissue-specific mechanical adaptations reflect differing patterns of gene expression at different body sites.

[‡] Upon receiving an antigenic stimulus, naive T helper lymphocytes differentiate into two distinct subsets – T helper 1 (Th1) and T helper 2 (Th2) cells. These subsets are defined by both function and cytokine profile.

Th1 pathway produces interleukin-2, interferon-γ, and tumor necrosis factor-β, which activate proinflammatory (M1) macrophages and initiate a complement cascade. This pathway is associated with transplant rejection.

The *Th2 pathway* produces cytokines that do not activate macrophages (namely IL-4, IL-5, IL-6, and IL-10). Th2 lymphocyte response releases noncomplement fixing antibodies, and is associated with transplant acceptance, mediated by the M2 macrophages.

Table 10.3 A brief summary of the main ECM proteins (A linear polymer of amino acids joined by peptide bonds in a specific sequence)

Protein type	Relevant biochemical properties	Main physiological roles	Disease associations
Fibrillar collagens			
I	Collagen type I is the most abundant ECM (>90% of dry weight), forming the white fibers of connective tissue. Molecular conformation is complex: • At the *monomeric* level, three polypeptide strands with a high glycine and proline/hydroxyproline content form a single left-handed helix. Fibrils are approximately 300 nm long and 1.5 nm in diameter. • At the *aggregate* level, three of these individual left-handed single helices twist into a right-handed triple helix, stabilized by numerous intermolecular hydrogen bonds. Vitamin C is an essential cofactor for the enzymes that stabilize the triple helix.	Provides tensile strength to bones, cartilage, tendons, muscle fibrils, soft tissue adventitia, dermis, and blood vessels. Within connective tissue ECM, a collagen type I scaffold supports and stabilizes the ECM as a whole. This framework also provides attachment sites for adhesive proteins and GAGs, and interacts with cytoskeleton to maintain “dynamic reciprocity” between the cells and the matrix.	Autoimmune disorders Osteogenesis imperfecta Ehlers Danlos syndrome Benign joint hypermobility syndromes Scurvy Lytic bacterial infections A 2-D polypropylene sheet can provide more than adequate tensile strength, but cannot replicate other features of a normal ECM.
III	Collagen type III is immature collagen, with a smaller fibril diameter and lower tensile strength than collagen I. It is the main collagen in granulation tissue and early wound healing. In contrast to the toughness required of a tendon or ligament, visceral parenchymas are typically embedded onto more flexible and compliant submucosae (e.g., liver, bladder wall). Collagen type III forms the reticular fibers that underlie such submucosae.	A high collagen III: I ratio is normal during early wound healing, but the ratio should reverse within 6 months – as newly formed collagen I fibrils dimerize with pre-existing collagen III molecules to form stronger triple helices. Failure of this ratio to reverse denotes a reduced proportion of high tensile strength (type I) collagen and an excess of immature (type III) collagen. Such a pattern suggests a collagen disorder.	Persistently high collagen III: I ratios (beyond 6 months) suggest a constitutional or acquired collagen disorder and abnormal fibroblast function.
Anchoring collagens			
IV	In collagen type IV, large segments of each individual triple helix splay apart into flat, sheet-like arrays. The NH_2 terminals of four individual triple helices then intertwine “head to tail”, forming a spider-shaped molecule (so named because it has a pair of splayed out “legs” projecting from each side of a small central “thorax”). Multiple “spiders” then join “foot to foot” at their COOH terminals, to form a honeycomb-like network. This unique stereochemical interaction of numerous collagen type IV tetramers creates a very strong skeleton, which entraps other molecules (laminin and entactin), to form a platform for cellular attachment.	In light microscopy, the stained structure that anchors an epithelial layer is known as the *basement membrane*. With the greater resolution of the electron microscope, the basement membrane typically encompasses two distinct layers – the *basal lamina* (secreted by epithelial cells) and a *reticular lamina* (secreted by fibroblasts). The basal lamina consists of an electron-lucid sublayer of laminin and fibronectin (for secure adhesion sites), overlying an electron-dense sublayer of type IV collagen (for tensile strength).	Thin glomerular basement membrane nephropathy (Goodpasture and Alport syndromes). The inability of a 2-D polypropylene sheet to form an effective basement membrane at the lamina propria / vaginal epithelial interface predisposes to mesh exposure and erosion.

Table 10.3 (continued)

Protein type	Relevant biochemical properties	Main physiological roles	Disease associations
VII	Individual collagen VII molecules have a unique shape: • The COOH terminal of the triple helix splays apart to form three "sticky fingers." • The majority of the molecule intertwines at the NH_2 end, forming a tight helix. The amino-terminals of multiple collagen molecules then aggregate into antiparallel dimers, with "sticky fingers" COOH terminals projecting from each end. Multiple dimers then aggregate to form anchoring fibrils, with a large bunch of "sticky fingers" at each end. These carboxyl-terminal ligands strongly bind keratinocytes to the basement membrane.	Collagen type VII anchoring fibrils at the dermal–epidermal junction protect keratinocytes from being pulled off the basement membrane by twisting or shearing forces.	Subepidermal autoimmune bullous disorders. Synthetic mesh erosions.
VI	The collagen type VI molecule consists of a short triple helical ending in prominent globular domains. Multiple dimers aggregate by antiparallel association, to form short helical filaments, interspersed by "sticky" beads.	Type VI collagen is the main connecting unit binding glycosaminoglycans to the structural protein framework (especially collagen type I) This association stabilizes the GAG-attracted water molecules within the ECM, forming a stiff gel.	Bethlem myopathy. Ullrich congenital muscular dystrophy.
Noncollagenous adhesive proteins			
Fibronectin	Fibronectins are high-molecular weight glycoproteins that exists in *soluble* (plasma globulin produced by hepatocytes) and *insoluble* forms (matrix adhesive proteins secreted by fibroblasts).	Fibronectins are the major "*structural*" ligands within connective tissue ECMs. Structural ligands: • Bind to fibrillar collagens • Collagen molecules, thus stabilizing the overall scaffold structure of the ECM.	Essential for embryonic mesodermal, neural, and vascular development. Major role in wound healing.
	The ECM (insoluble) fibronectins are linear poltpeptides, containing several tightly folded receptor sites. Two fibronectin polypeptides then associate into "handle bar" shaped diamers, linked by disulfide bonds. This association creates a series of paired ligand binding domains, spaced at strategic points along the "handle bar" shaped diamer. Specific regions act as high affinity binding sites for cell membrane receptors (integrins), collagen IV and VII molecules (basal lamina of basement membrane), and various components of the ECM ground substance (sulfated proteoglycans like heparan sulfate and fibrillar collagens).	• Attaches various cell types to the interior of the ECM interstitium. • Stabilizes the *basal lamina* to the collagen IV and VII molecules of the *reticular lamina* of basement membrane, thus counteracting shearing stresses on the epithelial cells. In addition to being a "structural" protein, fibronectin has several "*functional*" roles. • By linking the integrins to the sulfated proteoglycans, fibronectin is integral the cell-to-matrix cross talk that controls cell growth, migration, and differentiation. • Fibronectin also assists collagen type VII in binding hyaluronan, thus further stabilizing the gel properties of interstitial fluid.	Altered fibronectin organization is important in cancer metastasis and pathological fibrosis. The inability of synthetic and denatured biological mesh to induce normal fibronectin formation adversely impacts tissue turgor, cell cycle control, and homeostasis in the healed wound.

(continued)

Table 10.3 (continued)

Protein type	Relevant biochemical properties	Main physiological roles	Disease associations
Laminin	Laminins are a family of glycoproteins secreted by fibroblasts. They are the major noncollagenous component of the basal lamina. The laminin molecule has four arms, meaning that it can bind to four adjacent molecules. The three shorter arms are particularly good at binding other laminin molecules, forming strong flat sheets. The long arm is free to bind epithelial or endothelial cells, anchoring them to the underlying laminin-collagen IV platform.	Provision of a platform for cell attachment is a basic evolutionary requirement for multicellular organisms. The basic platform in mammalian ECMs consists of an aggregation of laminins, type IV collagen, entactin, and sulfated proteoglyans. • In their role as “structural” proteins, laminins bind the parenchymal cells to their supportive substrate. • In their role as “functional” proteins. the interaction of laminins with integrins in the plasma membrane helps control cell behavior and survival.	Dysfunctional structure of one particular laminin causes one form of congenital muscular dystrophy. Epithelial erosions overlying synthetic mesh implants.
Integrin	Integrins are a superfamily of cell surface glycoproteins,which span the *plasma membrane*. Integrins are medium-sized molecules that exist in a heterodimeric configuration (with a long α and short β subunit). • The base of the heterodimer is coupled with the cytoskeleton. • The free ends of the α and β chains project through the plasma membrane, creating a high-affinity binding domain on the cell surface. Signal transduction is accomplished by activating a tyrosine kinase cascade.	Integrins are not simple hooks. Working in concert with soluble signaling factors, integrins provide a bidirectional link between the cellular genome and the extracellular matrix. This messaging system tells the cell about the nature of its surroundings. Specifically: • Integrin messaging helps the cell make critical decisions about attachment, migration, differentiation, division, or death. • Integrin messaging activates the cell cytoskeleton to allow cell movement (essential to chemotaxis and angiogenesis), and to improve the security of cell-to-cell and cell-to-scaffold adhesion. • Integrin messaging drives a feedback loop by which cells sense what forces are acting on the matrix, and respond by altering fibroblast gene expression patterns to better suit their mechanical environment. Hence, integrins are a key factor in constructive remodeling and collagen homeostasis.	Tumor cell invasion and metastasis. Thrombotic infarction (CAD and stroke). Diabetic retinopathy. Epidermolysis bullosa and desquamative enteropathy. The inability of synthetic and denatured biological mesh to form adhesive proteins or integrins impacts cell cycle control and wound homeostasis. Conversely, a properly tensioned second-generation graft will undergo “smart” remodeling under the tutelage of ECM integrins - a process known as mechanotransduction.

Glycoeaminoglycans

Glycosoaminoglycans (GAGs) are pure carbohydrates, organized into long unbranched polysaccharide chains (with molecular weights of 10^5–10^7).

GAGs have two striking biochemical characteristics:

- In exact contrast to proteins, GAGs are brittle molecules (like celery), which provide only limited tensile strength to the tissues. Their straight and nonfoldable structure means that even a small mass of these long chain polysaccharide molecules will occupy a large tissue volume (Fig. 10.4a).
- GAGs have multiple sulfate and carboxyl groups, which surround the molecule with a large field of negatively charged radicals. This negative charge attracts a cloud of cations (such as sodium), which in turn draws in a great deal of tissue water (Fig. 10.4b).

Being low-density molecules with a high osmotic effect, GAGs impart high viscosity to the extracellular fluid. These

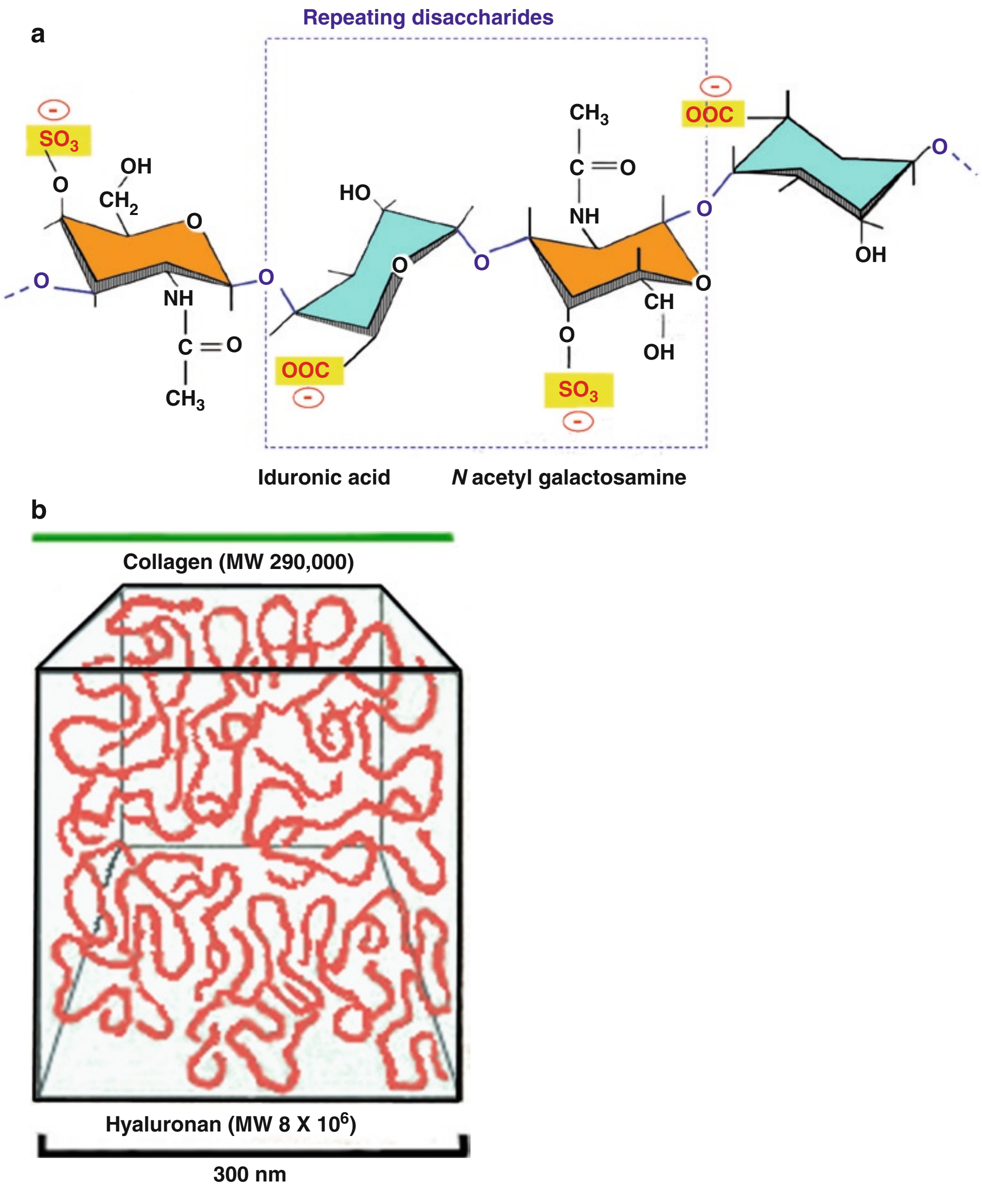

Fig. 10.4 Glycosoaminoglycans. (**a**) Glycosoaminoglycans have a very simple and infinitely repetitive molecular organization, consisting of hundreds (or even thousands) of disaccharide monomers, linked through oxygen molecules. The GAG disaccharide unit always consists of a uronic acid and an acetylated monosaccharide. The example illustrated here is dermatan sulfate. Multiple carboxyl groups on the iduronic acid and multiple sulfate groups on the *N*-acetyl-galactosamine create a large excess of negatively charged radicals. This negative charges attract a cloud of cations (such as sodium), which in turn draws in a great deal of tissue water. (**b**) Comparison of collagen and hyaluronan molecules. Collagen type I molecule has a compact linear configuration. Being electron neutral, it does not attract a surrounding layer of interstitial fluid. In contrast, hyaluronan forms extremely long, "non" folding chains, which occupy a large tissue volume.

turgid gels fill the interstitial space, maintain the viscoelastic properties of the ECM, and act as a compression buffer against shearing stress. As such, GAGs have quiet opposite physical properties to protein; however, the two together make a good partnership – one providing the necessary tensile strength and the other producing enough tissue turgor to resist crushing. Optimal wound healing in the vaginal walls requires that *neither* class of macromolecule be lost.

Structural polysaccharides in mammalian ECMs fall into two groups – sulfated and nonsulfated. The main nonsulfated GAG is hyaluronic acid, which is unique in that it does not combine with a core protein to form a proteoglycan. Hyaluronic acid polymers grow into very large molecules that bind a large volume of water. The main sulfated GAGs are heparin, heparan sulfate, and the chondroitin sulfates (Table 10.4). Sulfated GAGs exist mainly as protein conjugates (i.e., proteoglycans), rather than as unbound GAGs. Although they contribute somewhat to the gel properties of the ECM, the main role of proteoglycans is as regulatory molecules (see Chap. 10, *Bioactive Scaffold with Cell Adhesion Sites for Both Matrix Cells and Surface Epithelium* and *The Host's Ability to Revascularize an Implant Is the Rate-Limiting Step in Healing*).

Table 10.4 A brief summary of the main structural polysaccharides in the ECM

Class	Relevant biochemical properties	Main physiological roles	Disease associations
Nonsulfated glycosaminoglycans (GAGs). GAGs are long unbranched biopolymers, made up of repeating disaccharide units. These repeating units are joined together by covalent bonds (like beads on a necklace). Each monomer consists of an acetylated monosaccharide[a] and an uronic acid.[b]			
Hyaluronan (hyaluronic acid or hyaluronate)	The disaccharide monomer in hyaluronan consists of *N-acetyl-glucosamine* and *glucuronic acid*. The absence of a sulfate group means that hyaluronan cannot form the covalent bond needed to link this GAG molecule to a core protein. Hyaluronan is thus unique in being unable to assemble into a proteoglycan. Instead, hyaluronan forms very long chains with high electronegativity, thus imbibing water into the matrix.	Hyaluronan is the body's most abundant structural polysaccharide, and is widely distributed throughout the ECMs of connective, epithelial, and neural tissue. This ubiquitous carbohydrate biopolymer was once considered as just a "goo" molecule. It is now known to be metabolically active, contributing to tissue hydrodynamics, cell movement/proliferation, and a number of cell surface receptor interactions. It is also important to normal wound healing.	Inflammation and wound healing Osteoarthritis Ocular hypotonus Tumor cell invasion and metastasis.
Sulfated glycosaminoglycans and proteoglycans. Sulfated GAGs in the ECM are found mainly in association with either cell surface or ECM scaffold proteins, with which they assemble into proteoglycans (PGs). Multiple GAG molecules link to a single core protein through a serine-tetrasaccharide moiety (like bristles on a brush). Molecular shape and length vary enormously. Stereochemically, the basic brush-like construction undergoes complex tertiary folding, to better fit the diverse signaling and regulatory roles that PGs must fulfill. The detailed biology of proteoglycans is beyond the scope of this review.			
Chondroitin sulfates	The disaccharide monomer of chondroitin sulfates and hyaluronan both contain *glucuronic acid,* but chondroitins differ in having *N-acetyl-galactosamine*-sulfate as the monosaccharide. However, the chondroitin family is a heterogenous group; individual isoforms have quite diverse biochemical properties. A chondroitin chain can have over 100 individual sugars, each of which can be sulfated at different positions and in variable quantities.	Chondroitin sulfate PGs are a major component of the extracellular matrix. Of particular structural importance, chondroitin sulfates form a family of large aggregating proteoglycans (collectively termed the *lecticans*). These various lecticans (e.g., versican, aggrecan, neurocan) contribute to the tensile strength and compression resistance in *cartilage*, bone, tendons, ligaments, heart valve, and aortic wall connective tissue.	Mucopolysaccharidosis disorders Bone and joint diseases Cardiovascular disease
Dermatan sulfates	Dermatan sulfate was previously known as "chondroitin sulfate B." The dermatan monomer has the same monosaccharide (*N-acetyl-galactosamine*-sulfate) as the chondroitins, but differs in having *L-iduronate* as the uronic acid.	Dermatan sulfate proteoglycans fulfill important functions in dermal, vascular, and heart valve ECMs.	Mucopolysaccharidosis disorders Coagulation disorders Cardiovascular disease Carcinogenesis Infection Wound repair and fibrosis.
Keratan sulfates	The disaccharide monomer of keratan sulfate also contains *N-acetyl-galactosamine*-sulfate, but differs from the two previously described GAGs in having *galactose* as the uronic acid.	Keratan sulfates are large, highly hydrated molecules which create the light refractive properties of the cornea. Noncorneal isoforms act as a cushion to absorb *mechanical shock* in integumentary tissues (notably in *cartilage* and *bone*).	Mucopolysaccharidosis disorders Rheumatoid arthritis Alzheimer's disease
Heparan sulfates	The disaccharide monomer of heparan sulfate (HS) and heparin differ from hyaluronan in that both the main monosaccharide (*N-sulfo-glucosamine*) and the main uronic acid (*glucuronic acid*) are often sulfated.	Heparan sulfate is found in all animal tissues, mainly as an extracellular *proteoglycan*. HS-PGs regulate a wide variety of biological activities, including developmental processes, *angiogenesis*, *blood coagulation*, and tumor *metastasis*.	Mucopolysaccharidosis disorders Coagulation disorders Cardiovascular disease Carcinogenesis Amyloid precursor protein in Alzheimer's disease
Heparin	The repeating disaccharide units in heparin are similar to those in heparan sulfate, but there is a higher content of iduronic acid. The main biochemical difference is that heparin is about twice as sulfated, creating the highest negative charge density of any known biological molecule. Another fundamental difference is that heparin is an intracellular GAG, whereas *HS* is found mainly in the extracellular matrix.	Heparin is stored within the secretory granules of mast cells and released only into the vasculature or extracellular space at sites of tissue injury. Contrary to its medical use as an anticoagulant, heparin's main physiological role is as a defensive mechanism against invading bacteria and other foreign material at sites of tissue injury.	Mucopolysaccharidosis disorders Coagulation disorders Amyloid precursor protein in Alzheimer's disease

[a] *Acetylation* of a monosaccharide means the substitution of an acyl group [R–C(=O)] for one of the active hydrogen molecules in a simple sugar. The carbon of the carbonyl radical has an uncommitted electron available, through which it can bond to other molecules

[b] *Uronic acid* is a sugar in which the hydroxyl radical on the terminal carbon has been oxidized, forming a carboxylic acid

Growth Factors

Besides providing structural support, ECMs also serve as a reservoir of growth factors[§] and cytokines.[¶] These powerful bioactive molecules can function as oncogenes; hence, the ECMs must carefully modulate their synthesis and degradation. Cytokines and growth factors of most importance in connective tissue ECMs are basic fibroblast growth factor (bFGF), transforming growth factor beta (TGF-beta), vascular epithelial factor (VEGF), platelet-derived growth factor (PDGF), and keratinocyte growth factor (KGF) (Table 10.5). These factors tend to exist in multiple isoforms, each with its specific biological activity.[8]

Purified forms of growth factors and bioactive peptides have been investigated as independent therapies, to either encourage (VEGF) or retard (angiostatin) blood vessel formation, to stimulate deposition of granulation tissue (PDGF), and to induce re-epithelialization of wounds (KGF). However, such treatments struggle to deliver the right dose to the right site at the right time.[8] One of the major advantages of using a natural ECM as a scaffold for tissue repair is that the necessary growth factors (and their inhibitors) are present in appropriate amounts, still organized in their native three-dimensional ultrastructure.

Biochemical Principles That Dictate Surgical Outcome

The ECM is a vital, dynamic and indispensable component of all tissues and organs. It is nature's natural scaffold for organ morphogenesis, tissue homeostasis and reconstruction following injury. The degree to which a surgeon can preserve normal ECM structure and function has a profound effect on final wound quality.

Macromolecules and Matrix Cells Must Be Widely Dispersed Within a Generous Extracellular Space

As stated, integumentary ECMs consist of 10% matrix cells and 90% intercellular space. Scar tissue is the exact obverse of normal connective tissue:

- Biochemically, scar tissue lacks elastin, proteoglycans, and a GAG layer. This is why scars feel so dense and inelastic.
- Histologically, scar tissue consists of a dense fibrous whorl of randomly oriented collagen bundles within a compact avascular matrix. That is to say, the altered feel to scar tissue is reflected in its architecture.
- Biomechanically, the loss of this longitudinal orientation (parallel to the lines of stress) means that a scar is about one-third less strong than the connective tissue it replaces.

All in all, surgeons have tended to overestimate the reparative value of nonreinforced native scar. *Surgical rule # 1* is that:

> *Irregularly oriented fiber bundles have a potential to stretch, meaning that scar tissue is not well designed to resist chronic load. Hence, it would be logical to bolster all prolapse repairs, even primary cases. However, use of a tissue augmentation material is particularly important for recurrent and high-risk cases, given the acquired connective tissue degeneration that often arises secondary to longstanding disruption of collagen homeostasis.*[49]

Unfortunately, scars do not have a functioning ECM, meaning that the "sponginess" of the vaginal wall may be lost if such bolstering is done with synthetic mesh.

ECM Relies on Fibrous Structural Proteins for Tensile Strength

Collagen type I is the dominant element in connective tissue support, forming the white fibers of connective tissue. The key physical property of collagen type I is a pattern of intra- and intermolecular hydrogen bonds that confer enormous tensile strength. Unfortunately, the collagen laid down after wounding is invariably weaker than what it replaces, due to reduced fibril diameter and altered fiber orientation. Incorporation of synthetic mesh can certainly strengthen such scars. *Surgical rule # 2* is that:

> *Adding of an internal latticework of plastic mesh will always improve a scar's resistance to stretch under chronic load. Such increase in scar tensile strength will more than compensate any collagen weakness arising from metabolic degeneration within a chronically displaced cystocele or rectocele.*

As a corollary, it has been said that:

> *If you want a permanent result, use a permanent material.*

[§] Growth factors and cytokines are both signaling proteins that exert growth modulating actions.

Growth factors are small proteins that bind to cell surface receptors, with the almost exclusive result of activating cellular proliferation and/or differentiation (rather than pro-inflammatory effects).

[¶] *Cytokines* are small signaling proteins that regulate the behavior of other cells, especially hematopoietic and immune cells. Although many cytokines exhibit growth factor activity, their modes of action most resemble hormones – even to the extent of exerting autocrine, paracrine, and endocrine effects. The term tends to be used as a convenient generic shorthand for proinflammatory proteins such as interleukins, lymphokines, TNF and interferons.

Table 10.5 A brief summary of the main ECM growth factors and cytokine groups

Protein type	Relevant biochemical properties	Main physiological roles	Disease associations
Growth factors			
Growth factors are proteins that bind to receptors on the cell surface, with the primary result of stimulating cellular proliferation and/or differentiation.			
Fibroblast Growth Factor (b-FGF)	FGFs are important signaling molecules that bind to surface receptors on the plasma membrane, activating tyrosine kinase (TK) as a second messenger system. A TK cascade phosphorylates adaptor proteins within the intermembrane space, which activates intracellular effector proteins. These effector proteins then travel to the nucleus, where they can alter gene expression. Downstream cellular responses help regulate cell division, differentiation, and morphogenesis. • FGFs exist primarily as a family of soluble heparin-binding polypeptides, secreted into the interstitial fluid by local parenchymal cells. • However, there is a second pool of FGFs within the ECM, loosely adherent to heparan-sulfated proteoglycans. This phenomenon retains a crucial supply of FGFs at the cell surface, where they are needed for mechanotransduction signaling.	FGFs are pluripotent growth factors with mitogenic, morphogenic, regulatory, and endocrine effects. • During embryogenesis, FGFs are essential to cardiovascular and skeletal development. They also induce *mesodermal differentiation*. This latter phenomenon is what allows fetal wounds to heal by tissue regeneration, rather than by scarring. • In adults, FGFs help control *angiogenesis* (especially endothelial cell proliferation) and *wound healing* (especially fibroblast and keratinocyte proliferation). Even in adults, wounding releases FGF into the ECM – but in amounts that are too small to initiate mesodermal differentiation. Constructive remodeling. Thus, adult tissues are left to heal by neovascularization and subsequent scarring organization of the initial blood clot. However, the presence of adequate FGF in a suitable xenograft can induce enough mesodermal differentiation to allow adult wounds to recapitulate fetal healing.	Craniosynostosis and chondrodysplasia syndromes. Cleft lip and palate. Congenital diaphragmatic hernia. Parkinson's disease. Essential to healing by constructive remodeling.
Epithelial Growth Factor (EGF or KGF)	EGF is a polypeptide secreted by mesenchymal cells, which acts as an epithelial mitogen. Receptor binding leads to signal transduction through the tyrosine kinase pathway. EGF is now believed to be part of the FGF family.	EGF has proliferative effects on both keratinocytes and fibroblasts. EGF is upregulated after epithelial injury, suggesting an important role in wound repair.	Tumor progression in breast, endometrial, and prostate carcinomas.
Transforming Growth Factor - beta (TGF-β)	TGF-β is a protein originally purified from a tumor cell line; scientific interest was aroused because TGF-β could induce a reversible neoplastic transformation of nonneoplastic cell cultures. Similar properties have since been recognized in the reactin and inhibin family of proteins. TGF-β and related polypeptides bind to specific receptors on a variety of cells, thereby initiating signal transduction through the serine/threonine kinase pathway.	TGF-β is expressed to some degree in all tissue, where it functions as both a growth factor and a growth inhibitor. It is a major regulator of the *cell cycle*. When a cell undergoes malignant transformation, parts of the TGF-β signaling pathway are mutated, such that TGF-β can no longer promote apoptosis or stop the cell cycle at the G1 stage. TGF-β also plays a role in embryogenesis, immunity, cancer, heart disease, and Marfan syndrome. In adult wound healing, the TGF-β3 isoform modulates host response toward a rapid orderly deposition of ECM components, and away from an M1 macrophage infiltrate and a pro-inflammatory cytokine profile.	Arteriosclerotic vascular disease Tumor progression in breast adenocarcinoma Scleroderma and cystic fibrosis Marfan syndrome TGF-β acts as a chemotactic factor in wound healing, stimulating granulation tissue and increasing wound strength. Deficiency reduces wound strength.

Platelet-Derived Growth Factor (PDGF)	PDGF is a dimeric glycoprotein, secreted by platelets, endothelial cells, and placenta. Receptor binding leads to signal transduction through the tyrosine kinase pathway.	PDGF is another growth factor that regulates cell growth and division. • During embryogenesis, PDGF induces mesodermal differentiation and is essential to normal vascular development. • In adults, PDGF promotes macrophage, fibroblast, smooth muscle, and epithelial cell division. In essence, PDGF allows fibroblasts to skip the G1 checkpoints during blood vessel formation. The *cis* oncogene is derived from the PDGF B-chain gene. Overexpression of this gene in cancers induces uncontrolled angiogenesis. PDGF also promotes adult wound healing. It is a required element in fibroblast cellular division, and also facilitates keratinocyte proliferation.	Arteriosclerotic vascular disease Tumor progression in breast and other adenocarcinomas Scleroderma and cystic fibrosis PDGF is also a chemotactic factor in healing wounds, stimulating both inflammatory cell migration and new ECM deposition.
Vascular Epithelial Growth Factor (VEGF)	VEGF is also a dimeric glycoprotein that creates signal transduction through the tyrosine kinase pathway.	VEGF is probably a subfamily of platelet-derived growth factors. Cellular secretion is induced by hypoxia. • During embryogenesis, VEGF induces both *vasculogenesis* (the *de novo* formation of the embryonic circulatory system) and *angiogenesis* (vasculature budding from pre-existing blood vessels). • In adults, VEGF stimulates endothelial cell mitogenesis and cell migration. VEGF is a vasodilator and increases microvascular permeability; hence it was originally referred to as "vascular permeability factor." In wound healing, this property is chemotactic for macrophages and granulocytes.	Overexpression of VEGF promotes metastasis, and has been shown to be an adverse prognostic factor in breast cancer. VEGF is also overexpressed in rheumatoid arthritis and inflammatory bowel disease. VEGF stimulates endothelial cell mitosis and motility during wound healing.
Cytokines *Cytokines*[a] *are signaling proteins involved in local cellular communication, especially during embryogenesis and in hematopoietic / immune function. Secreted primarily from leukocytes, cytokines stimulate both the humoral and cellular immune responses, and also activate phagocytic cells. Many cytokines exhibit growth factor activity; however, some exert an inhibitory effect on cell growth or proliferation – even to the extent of mediating cellular apoptosis.*			
Acute Inflammatory Response (IL-1, IL-6, IL-8, and TNF-α)	Interleukins and tumor necrosis factor-alpha are small helical proteins with activity-specific folded domains, which serve as inflammatory mediators. Collectively, these acute response-modifying interleukins increase T and B cell production, as well as releasing chemoattractant messages.	In immediate response to injury, local inflammatory cells (neutrophils and macrophages) release a number of cytokines into the bloodstream, most notable of which are IL-1, IL-6, and IL-8, and TNF-a. These cytokines then induce secretion of "acute-phase proteins" (like C-reactive protein and coagulation factors) from the liver.	Deregulation has been implicated in many disease processes, especially inflammatory and malignant disease.

(continued)

Table 10.5 (continued)

Protein type	Relevant biochemical properties	Main physiological roles	Disease associations
Transplant Rejection Response (IL-2, IL-12, INF-γ, and TNF-α)	Upon receiving an antigenic stimulus, naive T helper (T_h) lymphocytes differentiate into one of two distinct subsets. The *T_h1 pathway* produces interleukin-2, interferon-γ, and tumor necrosis factor-β, which promote pathogen killing and phagocytosis of any foreign material or cellular debris.	In wound healing, the T_h1 pathway activates the proinflammatory (M1) family of macrophages, leading inevitably to chronic inflammation and eventual scar formation.	This pathway initiates a complement cascade, and is associated with transplant rejection. In wound healing, these cytokines favor scar formation.
Transplant Acceptance Response (IL-4, IL-5, IL-6, IL-10 and IL-15)	The *T_h2 pathway* produces cytokines that activate immunomodulatory macrophages (IL-4, IL-5, IL-6, and IL-10,). This second phenotype of macrophages (signified as M2) promotes an immuno-regulatory response, culminating in tissue repair and constructive tissue remodeling.	In wound healing, the T_h2 pathway activates the M2 family of macrophages, which permit constructive remodeling of a xenograft with the necessary growth messages.	T_h2 lymphocyte response releases noncomplement fixing antibodies, and is associated with transplant acceptance.

[a] Cytokines are a large family of signaling proteins that are produced by various cells. Those secreted from lymphocytes are termed *lymphokines*, and those from monocytes or macrophages are termed *monokines*. A subset of lymphokines, called *interleukins* (*IL*), exhibits a dynamic reciprocity: they are secreted by hematopoietic cells, with which they then interact

This aphorism is true, but the surgeon should also remember that:

> *Any morbidity due to the alloplastic mesh will be equally permanent.*

Why is this so? There are two reasons. Alloplastic mesh itself is inherently inelastic and tissue flexibility is further reduced by the ~25% mesh contraction usually seen during the healing phase.[15,50] Hence, the use of polypropylene implants can make the healed repair even harder and stiffer than would be the case with natural scar fibrosis.

The quandary facing reconstructive surgeons is how to restore tensile strength without losing tissue flexibility or overwhelming the viscoelastic properties of the connective tissue ground substance. Searching for a less morbid bolster, manufacturers chemically altered various cadaveric and animal grafts, in the hope of getting an equally permanent but "more natural" scaffold. Although aldehyde or carbodiimide cross-linking can create desirable mechanical properties (e.g., in extending the life of transplanted porcine heart valves), use of a biologically interactive scaffold material for prolapse surgery is seldom a good option. *Surgical rule # 3* is that:

> *Scientifically speaking, first-generation biomesh are no longer considered a suitable choice for primary wound support. Adulterated collagen scaffolds heal by encapsulation, and there is an ever present risk of late graft autolysis.*

Tissue engineering principles have taught us that the key determinant of whether downstream healing events follow an inflammatory or tissue inductive pathway is the presence or absence of a long-term foreign body in the wound.[28,42] *Surgical rule # 4* is that:

> *The most practical solution to mesh-related morbidity is to use an absorbable scaffold with remodeling properties.*

Obviously, the protein scaffold must be strong enough to splint the wound until host cell repopulation and graft remodeling are complete. Implant design must therefore counterbalance graft degradation and tissue induction rates (Fig. 10.5).

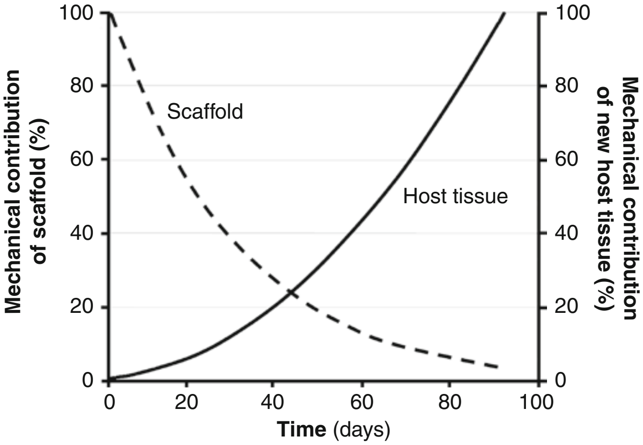

Fig. 10.5 Balancing speed of degradation against speed of remodeling. Whether temporary or permanent, the primary purpose of a surgically implanted "scaffold" is to ensure the mechanical integrity of the healing wound. This is easily accomplished with polypropylene mesh. But "constructive remodeling" is possible only if the implanted scaffold degrades quickly and remodels under site-appropriate mechanical loading. Tissue engineers must therefore design any biological implant such that the rate of scaffold degradation is counterbalanced by the rate of new tissue formation (Reproduced from Badylak et al.[30]. With permission from Elsevier)

Bioactive scaffold with cell adhesion sites for both matrix cells and surface epithelium

Skin wounds on early mammalian embryos heal perfectly with no scarring; in contrast, adult wounds invariably scar.[51,52] Much of this adverse healing pattern is attributable to disruption of the dynamic reciprocity between the cellular cytoskeleton and the adhesive proteins of the ECM (Table 10.3). Briefly:

- *Fibronectin*: Fibronectin is a high-molecular weight glycoprotein with two key "structural" actions. First, fibronectin conjugates into a handle bar-shaped dimer with high-affinity binding sites for *surface membrane receptors* (integrins) on fibroblasts, keratinocytes and endothelial cells. Second, fibronectin is also a powerful ligand for many *extracellular substances*, such as collagen type I (thus stabilizing the ECM cells to the fibrous framework) and site-specific GAGs (thus cementing the fibrous framework to the gel layer and regulatory proteoglycans). Without fibronectin, the extracellular matrix cannot properly balance tensile strength, elasticity and volume persistence.
- *Laminin* is a flattened, "cross shaped" molecule that complexes with nonfibrillar collagens (types IV and VII), integrin and entactin,[**] to form the basement membrane. Interactions between laminin and cell membrane receptors stimulate the actin filaments within the cytoskeleton[††] to contract, thus actively resisting surface shearing forces.[53] Without laminin, any overlying epithelium will be poorly adherent, as seen at sites of mesh erosion.
- *Integrin*: Integrins are small transmembrane glycoproteins that form a physical bridge between the microfilaments of the cytoskeleton and the macromolecules of the

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[**] *Entactin:* A dumbbell-shaped glycoprotein found in all basement membranes. It binds strongly to laminin and to collagen type IV.

[††] *Cytoskeleton:* A general term for an internal scaffolding in animal cells, which gives them structural strength and motility. The major components of the cytoskeleton are contractile microfilaments (actin), cell anchoring intermediate filaments (keratin), and compression-resisting microtubules.

> extracellular matrix.[54] The primary role of integrins is as "signaling molecules," passing messages in both directions.[53,54] *Matrix to cell messaging* transduces information from the external environment to the cell genome, altering gene expression to suit site-specific needs.[55] Outside-in signals empower cells to migrate through the ECM in response to chemotactic or angiogenic messages. Cytoskeleton activation also improves the security of cell binding, both at the basement membrane and within the matrix interior. Perhaps of most importance to the reconstructive surgeon, outside-in signals also drive the crucial process of mechanotransduction.‡‡ *Cell to matrix messaging* adapts the protein scaffold in keeping with any new patterns of gene expression. In particular, inside-out signals align fibroblasts with the direction of stretch, thus coordinating collagen fiber orientation. They also increase collagen I and decrease collagen III secretion, further improving wound strength.[55] All in all, transmembrane crosstalk between the extracellular matrix and the cytoskeleton plays a major role in controlling the cell cycle.

Two dimensional synthetic mesh does not induce the secretion of adhesive proteins. First-generation grafts do contain adhesive proteins, but these molecules are rendered nonfunctional by metallic salt precipitation. The resulting loss of fibronectin-laminin-integrin complexing sacrifices much of the matrix sophistication acquired during mammalian evolution. In contrast, natural-state ECM grafts contain an abundance of functioning laminin, fibronectin and integrin molecules, and can therefore serve as an information-rich prosthetic scaffold into which adjacent cells will migrate and remodel. *Surgical rule # 5* is that:

> *At sites where enduring tensile strength is the dominant consideration (e.g., sacrocolpopexy or midurethral slings), synthetic mesh may offer a genuine advantage. However, neither fibroplastic incorporation (of a monofilament mesh) nor fibrous encapsulation (of a chemically altered collagen graft) is conducive to forming a plump and flexible vaginal wall after prolapse repair. Preservation of full sexual function depends on more than just simple re-establishment of anatomic support. Only a tissue-inductive biomesh has the capacity to restore both normal anatomy and the dynamic reciprocity between laminin, fibronectin and integrins.*[49]

However, surgical use of remodeling biomesh is not as straightforward as it might appear. Healing response to a bioactive graft depends on the integrity of the collagen scaffold,[29] the source of cell repopulation,[21,56] the viability of xenogeneic signal molecules,[30] the forces present within the mechanical environment,[46,57] the rapidity of graft degradation,[28,41,58] and the host androgen status.[59-62] Overlaying a fistula closure with an interposition graft improves the likelihood of success; conversely, simply placing a natural ECM graft into the vesico- or rectovaginal space at colporrhaphy is not helpful. Under the dictates of tissue engineering theory, a simple onlay graft will remodel into additional loose fibrovascular tissue. Given that "thinning" of the endopelvic fascia is not an important factor in prolapse repair failure,[63,64] using an ECM biomesh in this way is both illogical and unsuccessful.[65,66] In contrast, suitably shaped and tensioned ECM grafts reduced early recurrence (a reasonable surrogate for technical failure) at vaginal paravaginal repair by 75%, and late recurrence (a reasonable surrogate for collagen weakness failure) by 67%.[49,67] In other words, a resorbable ECM graft can transform into a permanent and self-renewing layer of new aponeurotic tissue – but this requires some sophistication on the surgeon's part.

These clinical observations on SIS–ECM-augmented vaginal paravaginal repair are supported by considerable animal data. While direct graft repopulation from the immediately adjacent host cells is important, much of the cell recruitment actually comes from progenitor adult stem cells in the bone marrow[68,69] and surrounding blood vessels.[70] Remodeling into a site-appropriate tissue is as much influenced by mechanotransduction signals as by the actual cell source.[55] To study the effects of mechanotransduction on the remodeling process, rabbits were randomized to five different protocols.[57] The first control group had a sham operation where the Achilles tendon was exposed, but no defect was created and no SIS–ECM material was implanted. The other four groups had 1.5 cm of Achilles tendon removed from a hind limb. After tendon repair with an SIS–ECM interpositional graft, the limb that had been operated upon was immobilized for 2 weeks. One of the tendon repair groups was sacrificed at the end of this immobilization period, as additional controls. In the three surviving tendon repair groups, animals had the relevant stifle joint braced to allow full motion, partial motion, or no motion for an additional 4 weeks. All Achilles tendons were then harvested and examined microscopically. In the groups allowed partial or full motion, histology showed dense and well-organized collagenous connective tissue, oriented to the longitudinal axis of the tendon. The only difference between partial and full motion was that the latter group had more fibroblasts in the center of their remodeled grafts. Quite a different picture was seen in the group in which the stifle joint remained immobilized for the whole 6 weeks. The SIS–ECM graft showed incomplete replacement. Fibroblast ingrowth was limited to the periphery of the implant, and collagen deposition was both sparse and disorganized. In fact, new host connective tissue formation had not advanced much beyond what was seen in the second control group (tendon repaired, but sacrificed at 2 weeks). Confirmatory experiments were done with SIS–ECM repair of surgically created defect in a rabbit medial collateral knee ligament[71] and a canine abdominal wall model.[43] These preclinical studies all indicate that active loading of a remodeling ECM scaffold accelerates the formation of a robust, site-appropriate connective tissue. The process appears analogous to normal homeostatic renewal of fatigued connective tissue, organized in response to mechanical signals.[45,46] *Surgical rule # 6* is that:

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‡‡*Mechanotransduction* is the process by which mechanical loads placed on connective tissues alter gene expression in fibroblasts, thus adapting the ECM macromolecules to better fit their environment. This process is very important during bone remodeling and wound healing.

> *There is a well-documented mechanism by which a resorbable bioactive scaffold can lay down a permanent and self-renewing layer of site-appropriate host tissue, with no potential for mesh morbidity. Success depends upon anchoring the ECM-graft across a site-appropriate cell source and upon suitably tensioning the implant in four directions (to provide appropriate mechanical transduction signals to the neomatrix).*

Sufficient Tissue Hydration to Produce Lubrication, Tissue Turgor, and Cell Migration Lanes

As explained in Section Macromolecules, there are two families of structural polysaccharides in the ECM:

- Glycosoaminoglycans are pure carbohydrates, comprising of up to 25,000 repeating units of glucuronic acid and *N*-acetyl-glucosamine. The GAG most involved in providing sufficient osmotic force to maintain the integrity of the ground substance is hyaluronic acid (Fig. 10.4b).
- The sulfate-rich GAGs exist mainly as proteoglycans (i.e., the combination of a GAG side chain with a central protein core) (Fig. 10.4b). Proteoglycans are a very diverse group of molecules, which control many aspects of matrix architecture[71-73] (Table 10.4).

In their use of augmentation materials, surgeons have focused almost exclusively on increasing tensile strength within a repaired wound. In so doing, they often lose sight of the fact that preservation of an adequate GAG layer is equally important. The absence of GAG adhesion sites limits the degree to which a mesh-augmented wound can recapitulate normal connective tissue turgor; a poor GAG layer also impairs ongoing tissue homeostasis. While reducing mesh weight appears sensible, studies to date have not confirmed lightweight mesh to be less cicatrizing. The safer approach is to carefully follow the hernia principles as they relate to prolapse repair (see Section Sufficient Tissue Hydration to Produce Lubrication, Tissue Turgor, and Cell Migration Lanes). In static tissues (e.g., sacrocolpopexy or a midurethral sling), there is not usually enough friction at the graft–tissue interface for reduced tissue turgor to be a problem (see Section Host Response to Implantation of a Biomaterial). Conversely, if the implant is placed in dynamic tissues abutting a hollow viscus, any compromise in ECM gel properties will likely accentuate the abrasiveness of synthetic mesh.[74] *Surgical rule # 7* is that:

> *The clinical consequences of compliance mismatch vary according to the implantation site and the surgical objectives. When the basic objective is to construct a static "neoligament" (e.g., in resuspending an inverted vaginal vault), fibrous incorporation of a narrow strip of synthetic mesh is a rational option. In contrast, cystocele or rectocele repair needs a relatively broad "bridging graft", not a narrow neoligament. Such an implant must be strong, but not excessively so. The main considerations are retaining tissue flexibility and minimizing the risk of erosion or pain. Clearly, a natural ECM graft has some definite advantages over a synthetic mesh in this situation.*

A surgical dilemma can arise in situations where an ECM graft may genuinely lack the requisite tensile strength. For example, patients with a genetic collagen disorder are innately incapable of strong healing, even if perfect host cell remodeling were to occur. In other women, there can be an acquired collagen weakness, through disordered homeostasis, subnutrition, senescence, prolonged androgen deficiency, or the catabolic effects of NIDDM. In such circumstances, a surgeon must balance the potential morbidity of using a synthetic mesh against the increased risk of reparative failure if mesh is not used. Attempts to hide the polypropylene mesh beneath a sheet of chemically altered ECM graft has not been particularly, as shown by the high morbidity attending the Avaulta device (CR Bard Inc, Murray Hill, NJ).[75] Alternatively, natural biomeshes like Surgisis® (Cook Medical, Bloomington, IN) or Xenform® (Boston Scientific, Quincy, MA) have a proven capacity to ensure adequate tissue turgor and lubrication.

The Host's Ability to Revascularize an Implant Is the Rate-Limiting Step in Healing

Angiogenesis is necessary for nutrition of the repopulating cells, elimination of waste products, prevention of seroma, defense against infectious agents, and removal of xenograft metabolites. As such, the ability of a degradable scaffold to support new blood vessel growth is the usual rate-limiting step in graft assimilation.

Angiogenesis is a multistep process involving attachment, proliferation, migration, and differentiation of microvascular endothelial cells. Each of these steps is facilitated by preserving the intended ECM graft in its natural state.[76] In unassisted wound healing, vascular buds cannot sprout until they have first digested the obstructing blood clot with protease enzymes. However, a natural ECM graft has the advantage of retaining the spaces left by the animal blood vessels, thus offering readymade migration channels. Endothelial cell proliferation is initiated by fibroblast growth factor and the sulfated proteoglycans, creating solid buds that attach to the still viable collagen[44,77] and fibronectin fibers.[78] These sprouts "claw" their way into the matrix, driven by integrin messaging. Differentiation into a tubular channel with a smooth muscle wall occurs under the influence of vascular epithelial and platelet-derived growth factors.[79] Paradoxically, rapid scaffold degradation is also a powerful facilitator of angiogenesis – because degradation products of the parent ECM molecules serve as chemoattractants for endothelial cells.[41,80]

Natural xenogeneic grafts produce a favorable local microenvironment, with a very low potential for morbidity. Rapid scaffold absorption[29,42] and the absence of a foreign-body inflammatory response[28] avoid the potential for a severe cicatrizing host response. Grafts are also highly infection resistant, reflecting their ease of revascularization and the formation of antibiotic-like metabolites during scaffold degradation.[81,82] However, these grafts must be implanted into a well-vascularized body site, directly abutting a source of suitable host cells. They can be used safely on peritoneal surfaces, but adipose tissue must be avoided. Several sterile abscesses occurred when some eight layer SIS–ECM urethral slings were allowed to project beyond the external oblique aponeurosis and into the subcutaneous fat.[83] This phenomenon explains why a remodeling xenograft should not be placed in the ischio-anal fossa. The only other SIS–ECM problem has been very occasional seroma formation, especially with eight or ten layer orthopedic devices. As a precaution, heavy constructs should be punctured multiple times with a wide bore needle (e.g., an intravenous cannula). *Surgical rule # 8* is that:

> *Both short and long-term morbidity associated with second generation xenografts are is low, but poorly vascularized and adipose sites should be avoided. Heavy weight ECM grafts should be perforated at multiple sites with a cannula before insertion. As with any biomaterial, care should be taken to ensure a dry wound – if homeostasis is marginal, placement of a suction drain is a wise precaution.*

What Is “Tissue Engineering”?

Experience from tissue banks showed that cadaveric skin could be used as an early wound dressing in burn victims. However, such allogenic grafts were inevitably rejected after about 21 days, because of delayed host immune response. Conversely, banked bone grafts used in orthopedic surgery were not rejected; rather, these implants remodeled into new bone. These observations showed that host immune response is directed against the donor cells (in the epithelium), not the macromolecules (in the bone extracellular matrix).

From these observations, a new Science for fabricating tissues, called “Tissue Engineering/Regenerative Medicine,” has developed. This new discipline crosses the boundaries of surgery, cell biology, molecular biology, biomaterial engineering, computer-assisted design, robotics, microscopic imaging, and bioreactor design. In a nutshell, the field of tissue engineering/regenerative medicine applies the quantitative principles of engineering to the study of life sciences, with the ultimate objectives of controlling stem cell delivery and orchestrating whole-organ regeneration ex vivo (Fig. 10.6). Neither of these lofty goals is presently feasible. However, biomaterials scientists now understand extracellular matrix grafting quite well.

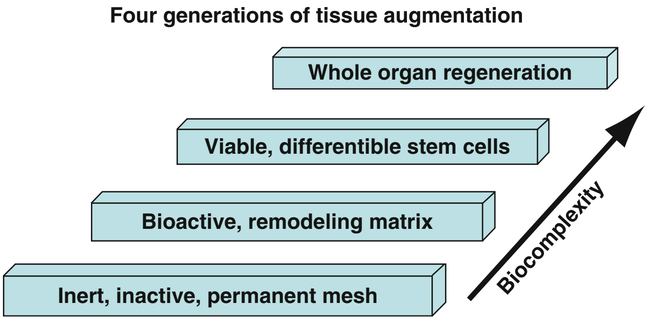
Four generations of tissue augmentation
Whole organ regeneration
Viable, differentible stem cells
Bioactive, remodeling matrix
Inert, inactive, permanent mesh
Biocomplexity

Fig. 10.6 Four generations of regenerative medicine. Intuitively, inert and nondegradable biomaterials appear ideal; but surgical reality is that the persistence of a permanent foreign body in the wound often proves disadvantageous. Skillfully used, a bioactive remodeling biomembrane can induce host formation of an equally permanent layer of strong new connective tissue – without risking wound morbidity. In that second-generation implants are acellular, a non-adulterated extracellular matrix is only “bioactive” (rather than “viable”). However, stem cell technology will indeed employ living and self-renewable pluripotent cells. In one sense, by wicking parietal fibroblasts from the obturator fascia to the pubocervical septum, VPVR with a Surgisis® bridging graft is already functioning at this third level of biocomplexity. The new science of tissue engineering aspires to one day being able to go to the fourth level, with an ability to generate a whole organ *in vitro*. Clearly, the biocomplexity increases as one goes from the second to the third and fourth generations of regenerative medicine (Reproduced from Hiles and Hodde [41]. With permission)

Extracellular Matrix Grafts

Tissue homeostasis is controlled by three components – the cells, the scaffold and the signals. With respect to wound repair, tissue engineering scientists asked a basic question: “Must all of these elements come from the host.”[40] Given that collagen is 99% preserved across all mammalian species, and can be transplanted from one species to another with minimal immunological response, the answer is obviously “no.”

Organs rich in parenchymal cells (such as kidney or liver) have very little ECM. In contrast, tissues with primarily structural functions (such as tendons, ligaments, pericardium, small intestinal submucosa, or dermis) have relatively large amounts of ECM. A wide variety of decellularized animal connective tissue have been studied, many of which are now commercially available. These ECM-grafts fit one or other of two broad patterns[8]:

- *Minimally altered collagen scaffolds*, prepared from structural tissues such as porcine small intestinal or urinary bladder submucosa, bovine pericardium and fetal

calf dermis. Such grafts comprise about 90% type I collagen, augmented by angiogenic factors and key matrix molecules. These grafts are primarily designed for orthopedic, body wall and prolapse surgery.
- *Basement membrane preparations*, prepared from such parenchymal tissues as porcine urinary bladder matrix or hepatic basement membrane. These grafts will eventually serve as a platform for stem cell implantation, and must therefore be rich in type IV collagen, laminin and fibronectin.

The most studied biomesh is porcine small intestinal submucosa, which is harvested from monitored pig herds and marketed as a 4-ply, lyophilized multilaminate called Surgisis® ES (Cook Medical Incorporated, Bloomington, IN). At the time of writing this chapter, Surgisis® ES is the only second generation xenograft licensed for use in Australia. Hence, all of my practical experience has been with this product. However, comparable products from other companies are available in other countries, as detailed in Table 10.2.

Host Immune Response to ECM Grafts

Transplantation of a whole-organ porcine or bovine xenograft into a primate would, of course, cause an immediate "hyperacute rejection." Such response is characterized by activation of the complement cascade, with thrombosis and graft necrosis in the implanted tissue. Hyperacute rejection is mediated by a powerful antigen called gal-(1,3)gal, found on the cell membranes of lower mammals (but not old world primates or humans).[84] Collagen itself has maintained a highly conserved amino acid sequence through the course of evolution, and is thus readily transplantable from one mammalian species to another. However, some of the other matrix molecules within these acellular collagen scaffolds still harbor residual gal antigens. Fortunately, these are sparse and exist in an isotopic form that does not induce complement-activating antibodies in humans.[58]

Are ECM Grafts Really "Tissue Inductive"?

Two decades of preclinical investigation has produced a wealth of highly reproducible cell culture and animal studies. Biomaterial scientists now have sufficient understanding of tissue engineering to guide surgeons in their rational use. A very brief selection of these studies is detailed below.

Early Vascular Studies: The site-specific remodeling capacity of ECM–SIS was first demonstrated in two landmark experiments, done at Purdue Engineering School in 1986–1991:

- In the first study, an autogenous ECM–SIS graft was prepared from a resected portion of the proximal jejunum, and used to replace a 5-cm segment of the infrarenal aorta.[85] One dog died with graft thrombosis 48 h after surgery. Nine dogs were sacrificed over a 52-week period; all grafts were patent, without adverse effects. The 100 μm SIS–ECM had remodeled into a 600 μm neoaorta that was histologically and functionally similar to normal aorta. Two dogs were allowed to survive and lived a healthy life for several years.
- In a follow-up study the next year, Badylak replaced the superior vena cava with another 100 μm tube of SIS–ECM in nine dogs.[86] Nine dogs were sacrificed at periodic intervals up till 72 weeks after surgery. All grafts were patent and had remodeled into normal, endothelium-lined vena cava. Two dogs were alive and well at 28 and 34 months.

The basic lesson from these paired experiments study is that the direction of SIS–ECM constructive remodeling was driven toward either aorta or vena cava, according to ambient intravascular pressure.

Reduced Esophageal Scarring Studies: The capacity of an ECM–SIS graft to heal by constructive remodeling (rather than scarring) was illustrated by patch grafting a 5 cm esophageal defect in a dog model.[87] Esophagus is notorious for severe stricture formation after any significant injury. Although the patch grafts did not remodel into completely normal esophageal tissue, they did heal into a functioning esophagus with a normal swallowing reflex. Histologically, there was preservation of three organized tissue layers, including good-quality skeletal muscle and a site-appropriate squamous epithelium. This result represents a dramatic improvement on the default healing response to esophageal trauma.

Body Wall Remodeling Studies: The constructive remodeling capacity of an ECM–SIS graft was demonstrated using a porcine urinary bladder matrix (UBM) scaffold to repair large full-thickness thoracic wall defects – including 5-cm segments of the sixth and seventh ribs.[88] The resected piece of the seventh rib was replaced as an interpositional bone graft, and buttressed with a UBM–ECM onlay. In contrast, the sixth rib defect was simply bridged with a sheet of UBM. In two control animals, similar defects were repaired with a Gore-Tex patch, which healed by mesh encapsulation within dense scar. Rather dramatically, all six of the UBM grafts remodeled into site-appropriate tissue, laying down organized fibrous connective tissue, muscle and new bone. In the sixth rib space, this new bone bridged the entire span. In the seventh rib space, new bone formation within the UBM implant had reunited the transected rib fragment to the adjacent rib ends.

In vitro Studies: A recent in vitro model showed that cyclic mechanical stretching of fibroblasts seeded on the SIS–ECM scaffold changed the gene expression pattern – increasing collagen type I and decreasing type III

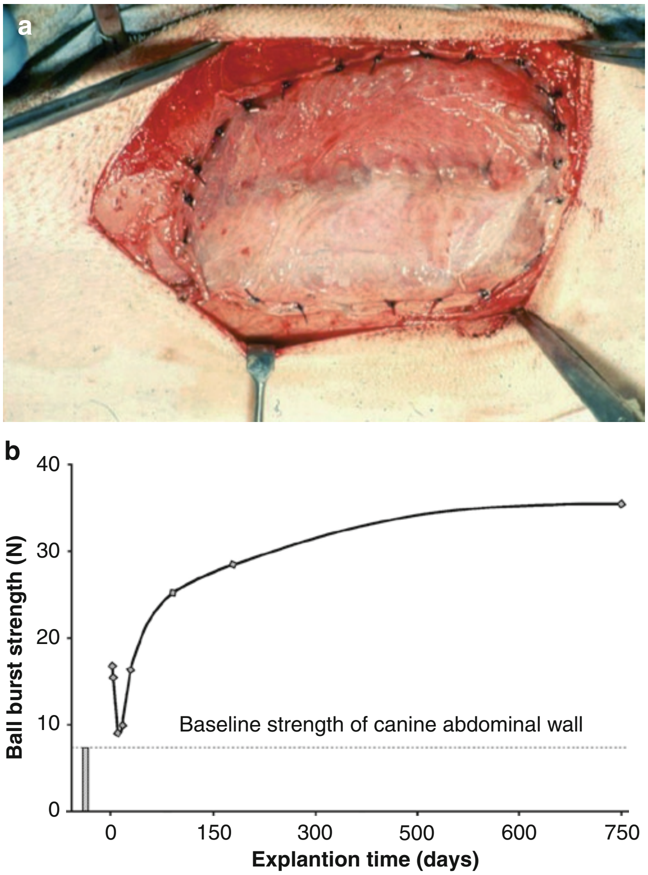

Fig. 10.7 Strength of remodeling xenografts over time. (**a**) As shown in this photograph, an 8 × 12 cm defect was created in the musculo-tendinous canine anterior wall, and replaced with an eight-layer SIS–ECM hernia device. The graft is much thinner than the tissue layers it replaces, as denoted by the semitransparent appearance. (**b**) The implanted SIS was initially twice as strong as the canine external oblique aponeurosis (N.T,). Serial strength measurements showed that the SIS–ECM weakened during the cell repopulation stage to about the same strength level as the native tissue, before remodeling to about five times stronger over the succeeding 2 years. This experiment has highlighted the need for ECM therapeutic scaffolds to be designed so that the rate of degradation is balanced by the rate of reconstruction, such that composite graft strength remains strong enough to splint the wound until healing is complete (Reproduced from Badylak et al.[4]. With permission from Elsevier)

production. These in vitro findings partially explain the improved healing strength produced by mechanical loading of the healing wound.

How Strong Are Remodeled ECM Grafts?

The final strength of a constructively remodeled ECM graft is greatly influenced by the site of implantation. To investigate the long-term healing strength in the abdominal wall, full-thickness 8 × 12 cm defects were created in 40 dogs (Fig. 10.7), and then repaired with an eight-layer SIS–ECM hernia device.[43] The load-bearing capabilities of the excised native tissue and the SIS hernia repair device were measured by ball burst tests,[§§] and compared to the composite strength of the excised device/implant site at the time of sacrifice. Preimplantation device strength was 16.5 ± 2.58 N (i.e., ×2.24 as strong as the excised native tissue). Load-bearing capacity at the surgical site almost halved at 10 days (8.99 ± 4.05 N), but was still ×1.22 as strong as the excised native tissue. Progressive remodeling of the implanted hernia device eventually doubled the abdominal wall strength (35.37 ± 5.86 N) over the succeeding 24 months, becoming ×4.59 as strong as the excised native tissue (Table 10.6).

Table 10.6 Mechanical property testing of explanted SIS hernia repair device using the Ball Burst Test

Survival time	Mean burst load		Standard deviation		Relative strength	
	Pound-force	Newtons	Pound-force	Newtons	Mean	SD
Native tissue	32.7	7.36	16.2	3.64	1.0	0.23
Implant-ready device	73.37	16.5	11.45	2.58	2.24	0.35
1 day	66.91	15.06	5.15	1.16	2.05	0.15
4 days	51.95*	11.69	7.49	1.68	1.58	0.27
1 week	42.77*	9.62	19.66	4.42	1.31	0.33
10 days	39.97*	8.99	18.03	4.05	1.22	0.55
1 month	72.65	16.35	38.12	8.57	2.22	1.17
3 months	109.63	24.67	63.06	14.19	3.35	1.93
6 months	120.72	27.16	39.47	8.88	3.69	1.21
2 years	157.20*	35.37	26.03	5.86	4.59	1.01

* *P*, 0.05

[§§] *Tissue strength* is measured by the force that the explanted grafts could withstand. Force means the interaction between molecules that causes an object to accelerate or deform. This phenomenon is best determined by a ball burst test, which applies perpendicular multidirectional force to the test material, using a constant-force compression cage. Ball burst test devices closely mimic the in vivo load experienced by the tissue. Force can be measured in "pounds-force" (Imperial units) or "Newtons" (SI units). 1 N is the force of Earth's gravity on an object with a mass of about 102 g (such as a small apple). At sea level, the downward gravitational force on a 70 kg man is approximately 687 N. Burst strength of Marlex mesh and SIS is comparable.[89]

Clinical Experience with ECM Grafts

Natural ECM have been successfully used for the reconstruction of numerous body tissues including musculo-tendinous structures, lower urinary tract, dura mater, vascular disorders, and body wall repair. In my personal series of vaginal paravaginal repairs for severe cystocele,[49,67,90] Cox proportional hazards modeling showed that the use of a suitable bridging graft reduced the relative risk of repair failure by an average of 69.4% (range = 26.9–86.9%). With respect to the time to failure, tissue augmentation reduced early recurrence (i.e., technical failure) by 75% and late recurrence (i.e., collagen weakness) by 67%.[49] However, a full review of this information is obviously beyond the scope of the present chapter.

Conclusion

There is a striking discordance in how information on ECM grafting has been received among engineers and scientists, as compared to surgeons. The development of tissue inductive therapeutic scaffolds has created a paradigm shift in Biomaterials Science; it has also attracted substantial investment from Industry. In contrast, 20 years of ingenious preclinical evaluation have gone virtually unnoticed in clinical circles. Prolapse surgeons have persisted in the use of unduly morbid polypropylene devices or obsolete cross-linked xenografts – not because of a conviction that these older materials are better, but because articles from the scientific literature do not achieve circulation among clinicians. However, given the medico-legal storm clouds gathering around the use of trocar-placed synthetic mesh for prolapse repair, there is an urgent need for surgeons to assimilate the knowledge attained through preclinical experiments.

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Part III

Anterior Defect Repair

Cystocele Repair with Mesh (Fixed Implant)

11

Emmanuel Delorme, Jean Pierre Spinosa, and Beat M. Riederer

Introduction: History

Prosthesis was first used to treat cystocele by abdominal surgery. In 1996, for the first time, a prosthesis was used by vaginal way with sutures fixation.[1] Petros and Ulstem invented tension-free suspension concept in 1993 with retropubic tape[2,3] and after that they developed the transgluteal posterior tape.[4] The transobturator tape was published to treat incontinence in 2001 with an evocation to use transobturator route to treat cystocele. The first publication of mesh repair with transobturator arms was in 2003.[5]

In order to understand the cystocele repair with fixed implant mesh, it is essential to have a good knowledge of the pelvic anatomy and a good approach of the structure and shape of the prosthesis. We developed a technique with six arms: two anterior transobturator arms (ATO), two posterior transobturator arms (PTO), and two posterior transsacrospinous arms (USS).

Surgical Anatomy of the Anterior Mesh

To understand the correct positioning of the prosthesis, a thorough knowledge of normal anatomy and of anatomical dangers is mandatory. The aim of the anterior mesh is to restore the function and anatomy of the bladder. We must remember that the bladder and the first third of the urethra are intrapelvic, meaning, above the Levator ani muscle. The lower third of the urethra is perineal, meaning, below the Levator Ani muscle. The middle third of the urethra is at the level of the Levator ani muscle. Surgery for prolapse is a pelvic surgery (above the muscle) and is different from surgery for incontinence (perineal). The region below the uretro-vesical junction classically must stay elastic and sliding, free from any scar risk, should it be prosthetic or surgical. The normal suspension system is composed of six ligaments. The meshes, the aim of which is to reinforce or replace the fascia pelvis, should ideally be secured by six arms: two anterior in the direction of the pubo-vesical ligaments, two lateral replacing the arcus tendineus fascia pelvis, and two posterior (utero-sacral ligaments). The anterior arms can be placed by the anterior obturator route, the lateral arms by the posterior obturator route, and the posterior arms can be placed “a retro” through the ischio-rectal fossa. Each one of these steps has anatomosurgical dangers that should be known. We shall describe the main risks for the anterior arms, the medial arms, and the posterior arms.

1. *As mentioned above, the placement of the anterior arms* is different from the placement of a tape to cure incontinence, as we have to stay voluntarily intrapelvic. The transobturator out–in allowed this step without particular dangers. Only vascular aberrations can be the source of important bleeding and this is a complication inherent to the anatomical variations. There are no troncular nerve injury risks. It is not known if the parauretral and/or the para urethrovesical junction nerves injury have a clinical repercussion.
2. *The medial arms* are similar to the insertion of the fascia pelvis on the Arcus Tendineus Fascia Pelvis (ATFP). The ATFP is not really strong and not often a palpable structure. The trajectory going from the lower part of the pubis to the sciatic spine can be mentally visualized. One of the problems is that in 20% of the patients, the Alcock canal (including mainly the Pudendal nerve) is more or less at 1.5 cm below the insertion of the ATFP. In case of a direct transfixion of the inferior part of the Obturator Foramen there is then a risk of injuring the Pudendal nerve.

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3. *The posterior fixation* can be done by positioning the two arms in the direction of the sacro-spinous ligament. Theoretically speaking, the paths could be anterior to the ligament, through the ligament, and posterior to the ligament. This last option must be ruled out as the sciatic nerve runs immediately behind the ligament and there is a high risk of injury. Two possibilities remain, transligamentous or preligamentous. Whatever the surgeon's choice might be, we must remember that there are two important nerves that should not be injured. One, above the ligament and the coccygeus muscle is the Levator Ani nerve. The second, below the ligament (sometimes through the ligament) and the Levator Ani muscle is the Pudendal nerve. Anatomical studies have shown that both these nerves never run more than 2 cm medial from the Ischial spine. These anatomical findings give us the key to this surgical step: passing medially more than 2 cm from the ischial spine. Intractable pain can appear in case of injury of the Pudendal nerve.[6,7]

Finally, it is possible to transfix the Ischiorectal Fossa (IRF) with a needle and advance up to or just before the SSL. The needle then perforates the ligament or the Coccygeus muscle just before the SSL. The inferior rectal pedicle is at risk of injury in the IRF.[8] At the level of the perineum the needle perforates the skin and the subcutaneous tissue. There is a constant nervous branch innervating the sphincter Ani, at risk of damage.[9] It is mandatory to stay at a minimum of 3 cm below the level of the anus to avoid the inferior rectal pedicle. There have been no studies published, evaluating the neurophysiological consequences of a wrong placing on the anal sphincter physiology and function.

Prosthesis and Device Grounding for Cystocele

A mesh is defined by the structure of the thread, the kind of knitting, and the shape of the prosthesis. At the beginning of our experience (1999) we used nonwoven polypropylene. Our experience and the global experience confirm that this type of mesh increases the risk of infection and erosion. Since 2005 we have used a thin polypropylene mesh (ABISS) (Fig. 11.1). It is a large porous knitted mesh with polypropylene thread of 80 η diameter which gives a minimum weight of 22 mg/m^2. The spread of the mesh must widely cover the defect between the two Levator muscles. But it seems to us that it must not be too wide, so as to let the pelvic muscle supple and not stiffen the dynamic pelvic floor.

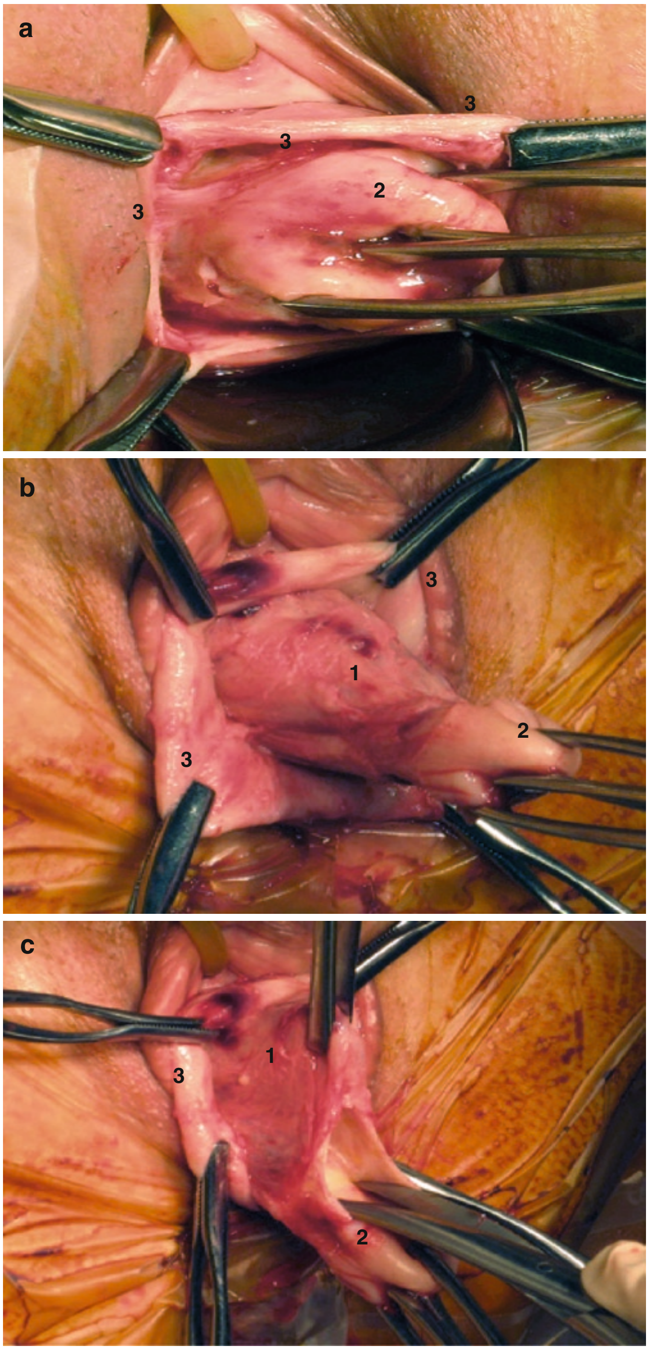

Fig. 11.1 Dissection (with uterus). (**a**) Incision. (**b**) To roll outside vagina wall. (**c**) Freeing bladder from the cervix. *1* Bladder, *2* Cervix, *3* Vaginal wall

It is difficult to evaluate the shrinking of the prosthesis. With empiric method, we decided that the size of the mesh would be 15% bigger than the defect. The length of the mesh is 9 cm, because the average length between the cervix and the bladder neck is about 7 cm.

The width of the mesh is 6 cm, because the average size between the two Levators muscles is about 4.5 cm. The arms are: anterior arms (ATO), lateral arms (PTO), and posterior arms. Posterior arms are transuterosacral and transsacrospinous (USS). This mesh is reinforced by a suture, which is knitted around the prosthesis (thread of polypropylene 150 η diameter). It gives little elasticity to the arms and allows a good traction to introduce them.

The devices to introduce the mesh are very simple. The more the device has a complicated shape, the more difficult it is for the surgeon to imagine the way inside the pelvis and the perineum. This is why we prefer only straight needles. We describe later the device with the technique, because the needle has the logical shape to simplify the technique.

Vaginal Incision and Dissection

The easiest incision is at the top of the prolapse, where the prolapse is outside the vaginal cavity. A short incision is better and as far away possible from the prosthesis.

1. *With the Uterus in Place and Prolapsed at Stage 2 or Higher* (Fig. 11.1)

The incision is made at 2 cm from the tip of the cervix. This incision must be deep opening mucosa and fascia. Laterally, in front of the uterus blood vessels, the incision must be more superficial. A strong pulling at the cervix opens the dissection plane: anteriorly between the bladder and the vaginal fascia, posteriorly successively between vaginal wall and peritoneum and after between vaginal wall and rectum. The vaginal wall is rolled outside "like the lid of a canned good." Rolling the vaginal wall and pulling the uterus give a good exposition of the dissection plan. The bladder is exposed up to the bladder neck if we want the mesh to stay up to it. Laterally to the bladder the tissue traction exposes the ATFP. This is the door to open the wide lateral bladder space, just behind the Obturator Fossa. This section of the ATFP must be done very carefully. Sometimes it is thin with no resistance, sometimes it is very stiff (young women, second time surgery). If the dissection of the ATFP is done badly there are two possible complications:

- Bladder injury: the injury will be at the level of the ureteral bladder ending. The repair needs endoscopic examination of the bladder and ureteral catheter (One case in our experience).
- Crossing the levator muscle. That can give heavy bleeding and needs hemostasis by abdominal or laparoscopic surgery (Two cases in our experience). To finish the bladder dissection, it is better to free the bladder from the uterus neck about 1 cm.

If there is no need to do a posterior repair, the posterior dissection can be limited. It is sufficient to reach the sciatic spine by a route just under the uterus pedicle, close to the Levator muscle and the pelvic wall, putting the finger in contact with the sciatic spine. A simple wiper movement from the sciatic spine to the sacrum frees the sacro-spinous ligament.

2. *Cystocele after Hysterectomy*

In this case, most frequently, the cystocele is accompanied by an anterior enterocele. The vaginal incision is sagittal, about 4 cm large, 3 cm above the bladder neck. It is a full thickness incision of the vaginal wall; the good plan is between the bladder and the fascia. It is possible to roll outside the vaginal wall to expose correctly the dissection plan, but it is not always necessary. The opening of the ATFP is the same as we described before.

Behind the bladder, the peritoneum is not opened but dissected from the vaginal wall to expose first the vaginal insertion of the uterosacral ligament (it is easy because in the vaginal cavity, there are two dimples at the insertion of the uterosacral ligament). Upwards, it is easy to follow the pelvic wall in order to reach the sciatic spine and free the sacro-spinous ligament. It is easy to reach the sacro-spinous ligament from the bladder dissection.

Technique to Introduce the Arms

The prosthesis is fixed by arms through the pelvic wall; anteriorly and laterally the Obturator muscles and posteriorly the sacro-spinous ligament. The good place of the prosthesis is determined by the good orientation of the arms. The ATO mimic the pubo-vesical ligaments, the lateral arms are under the Hypogastric vascular bundle and mimic the ATFP. The USS pass through the cervix insertion of the utero-sacral ligaments, behind they are lateral to the rectum and posterior through the sacro-spinous ligament very medial, lateral to the sacrum and very high. The sacral fixation of the utero-sacral ligaments is about 10 mm up to the upper corner of the sacral insertion of the sacro-spinous ligament. The posterior arms mimic the utero-sacral ligaments.

1. *The Way of the Anterior Arm is the Anterior Trans-obturator Route* (Fig. 11.2).

This route is quite different from the way of the T.O.T for incontinence treatment. The tape route in incontinence is a perineal way; the needle crosses the Obturator muscles focus on to the urethral meatus. The tape will be at the level of the middle third of the urethra.

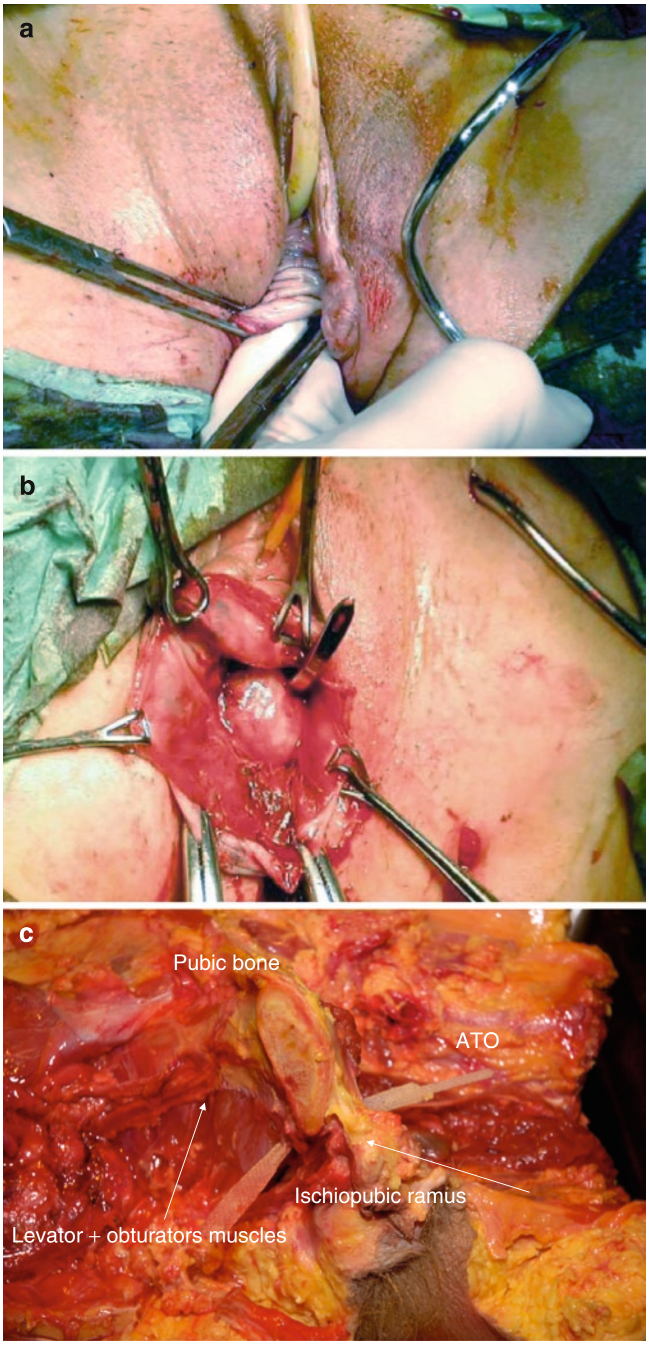

Fig. 11.2 The anterior transobturator arm. (**a**) Finger behind muscles to protect the bladder. (**b**) Dissection. (**c**) Pubic bone, ATO, ischiopubic ramus, levator and obturator muscles

In prolapse surgery the needle is introduced more vertically, focus upper to the bladder neck. That is possible only if the bladder is freed from the pelvic wall by section of the ATFP.

The classical Emmet needle small size with a blunt tip is probably the best tool for the anterior arm.

2. *The Medial Arm is the Posterior Transobturator Arm* (Fig. 11.3).

The way of this arm is probably the most difficult to understand. The needle is introduced posterior inside the Obturator Fossa, just close the Ilio-ischiatic bone. The Pudendal line is the line from the middle point of the posterior side (Ilio-ischiatique side) of the Obturator Fossa to the sciatic spine. The Pudendal nerve is always under and medial to this line, whatever the anatomic place of the nerve. The needle skin insertion point is just in front of the middle point of the Ilioischiatique side of the Obturator Fossa. The orientation of the needle is posterior and outside the Pudendal line. It crosses the Obturator muscle and it is between the Obturator and the Levator muscles, nearby posterioly the Ilio-pelvic bone and the Levator muscle. It passes through the Levator muscle 2 cm outside and at the sciatic spine level, never above the sciatic spine because there would be a risk to injure the sciatic nerve roots. It is not logical to turn the needle outside the vaginal incision because it would tear the muscles. It needs a device to grasp the arm on the tip of the needle (thread, tube, or other fixing systems), because no needle can have a shape adapted to turn at the level of the vaginal incision. The situation of the PTO is from the lateral side of the mesh to 2 cm outside the sciatic spine. This situation is safe for the Pudendal nerve and since the situation is away from the lateral sulcus of the vaginal vault, it lowers the shrinking and painful adherence to the vaginal wall.

3. *The Posterior Arm Has a Different Placement According to the Presence Or Absence of the Uterus.*
   - With the uterus present, the USS cross through the cervical insertion of the Uterosacral ligament (Fig. 11.4), which is very easy with an Emmet needle. Then the arms go lateral to the rectum and pass through the sacro-spinous ligament.
   - Without an uterus, the upper and posterior corners of the tape are sutured with a nonabsorbable thread to the vaginal insertions of the Utero-sacral ligaments and the USS pass lateral to the rectum and through the sacro-spinous ligaments.

The way through the sacro-spinous ligament and the buttock can be performed by the insertion of the needle from outside to inside or to inside to outside (Fig. 11.5). The OUT/IN way is classical (Ischio-anal way) and is described elsewhere in this book. Different needles are used, but the needle cannot turn up the vaginal incision. This is why it is necessary to have a fixing system on the needle to grasp the arm.

The IN/OUT way is done with a specific needle: the handle must be in the same axis as the needle to pass

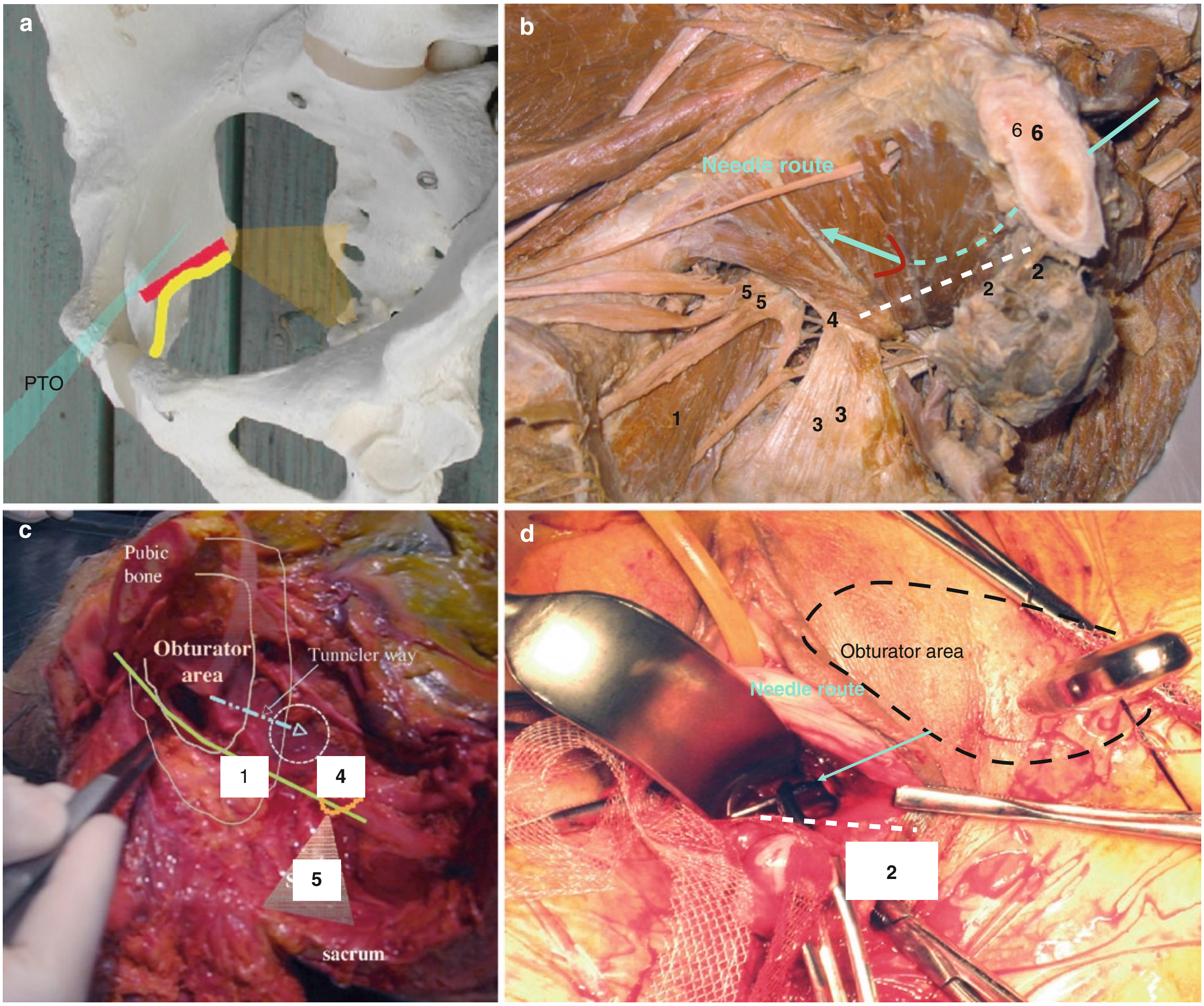

Fig. 11.3 The posterior transobturator arm (PTO): (**a**) PTO. (**b**) Dissection and needle route. (**c**) Dissection and obturator area. (**d**) Surgery. *1* Pudendal nerve, *2* Pudendal line, *3* Sacro-spinous ligament, *4* Sciatic spine, *5* Sciatic nerve, *6* Pubic bone

under the pubic bone. A non specific needle would limit the way.

The IN/OUT way is less dangerous than the OUT/IN way, because the needle comes from the dangerous area (sacro-spinous ligaments) to the nondangerous area (buttock). The way is shorter and does not reach the perineum where there are risks to injure any sensitive nerves. We used 50 times the IN/OUT way with no technical difficulty and good follow-up (no pain, no hematoma). The distance through the buttock is very short. At the end of surgery, it is better to free the arms before cutting the outside extremity of the arms. If you do not, the extremity of the arm would cross back the sacro-spinous ligament, and would not be strong enough to support the mesh. The sacro-spinous ligament IN/OUT way is a transbuttock way.

The best area to cross the sacro-spinous ligament is very medial, lateral to the sacrum, and very high on the ligament. That makes the way of the USS very close to the Uterosacral ligament way. The Utero-sacral ligament's sacral insertion is about 10 mm above to the sacro-spinous ligament, on the edge of the sacrum. In this situation, the axis of the vaginal cavity will be close to the normal axis (about 15° lower).

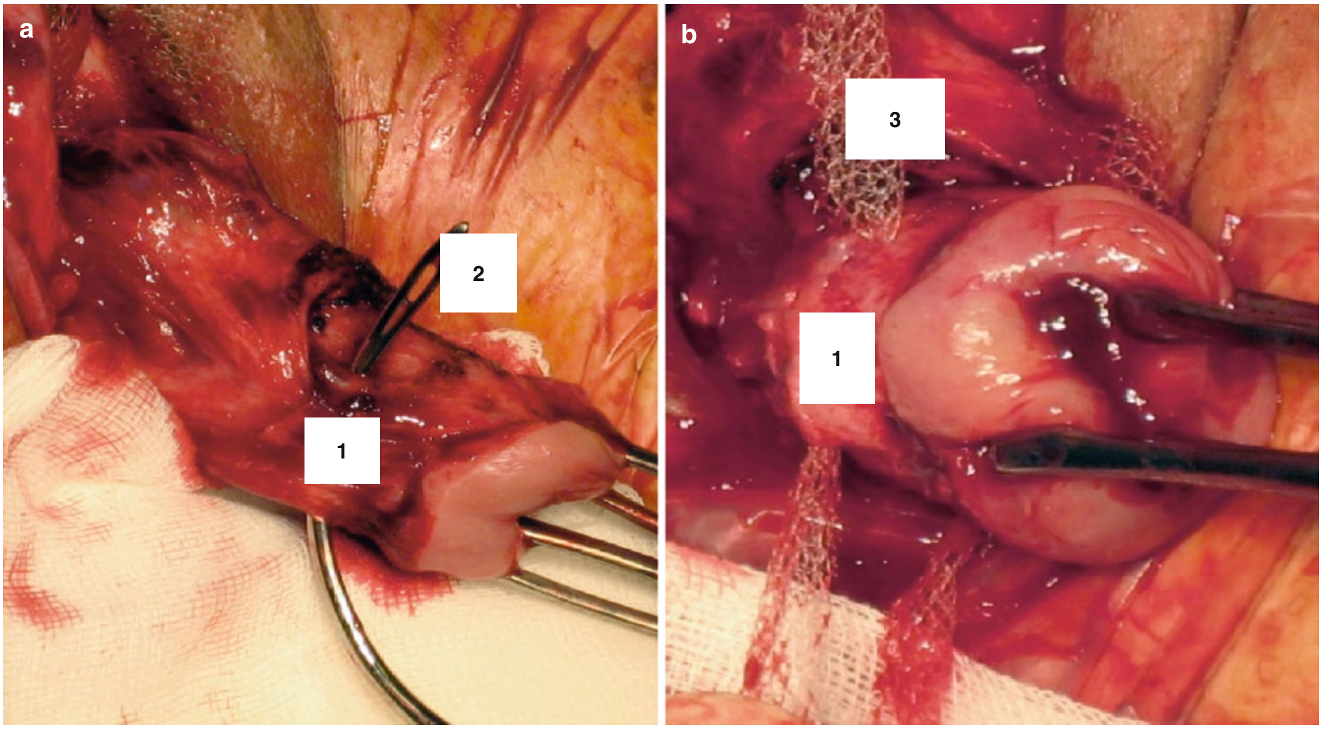

Fig. 11.4 The posterior arm: uterosacral ligament. (**a**) Passage of the needle. (**b**) Mesh in place. *1* Uteroscral ligament, *2* Needle, *3* Prosthesis arm

This area, inside the sacro-spinous ligament, is away from all dangerous structures (Pudendal nerve, posterior rectal nerve, roots of the sciatic nerve).

Drainage

Most of the complications after vaginal pelvic surgery come from occult bleeding and hematoma. Hematoma can be responsible for infection and perhaps prosthesis shrinking. This is why we decide to drain almost all the patients by a transobturator suction drain Chap. 12. The Emmet needle is inserted in the middle of the transobturator fossa. A drain is connected at the tip of the needle and removed through the Obturator Fossa. If the surgery is with low bleeding, the drain produces about 70 mL on the first postoperating day. If the surgery is with average bleeding, the drain may show up to 200 mL during the first postoperative day. In our experience the drain was never responsible for infection.

Vaginal Incision Closure

Closure of the vaginal incision must be very meticulous. Because the arms are not pulled, the incision is very low and easy to close with a good control. Our habit is to close the vaginal incision by two levels (fascia and mucosa) with absorbable continuous sutures.

Installation of the Prosthesis

The prosthesis is adjusted by pulling the different arms with no tension. The arms must be pulled in meticulous ship-shape. The ATO must be pulled first, not too strongly, just to spread out the prosthesis. It is important to check that the anterior side of the prosthesis does not strangle the bladder base up to the bladder-neck. Second, the USS must be pulled strongly, because they are the most important suspensive system of the mesh. The PTO will be pulled last with no tension. A special specific mention must be done about the lateral arm (PTO): the theoretical function of these arms is not to suspend the prosthesis. The tension of the mesh between the ATO arms and USS arms does not protect the prosthesis of a lateral shrinking. The ingrown fixation and the shrinking of the PTO arms would increase the lateral hold of the prosthesis and probably thus reduce the lateral shrinking of the mesh. Therefore the mesh would stay flat in a good place. We observe on an anatomic model that if we put tension on the PTO at first or second, the prosthesis is held with too much laxity. Thus, it is better to put the PTO under tension in the end.

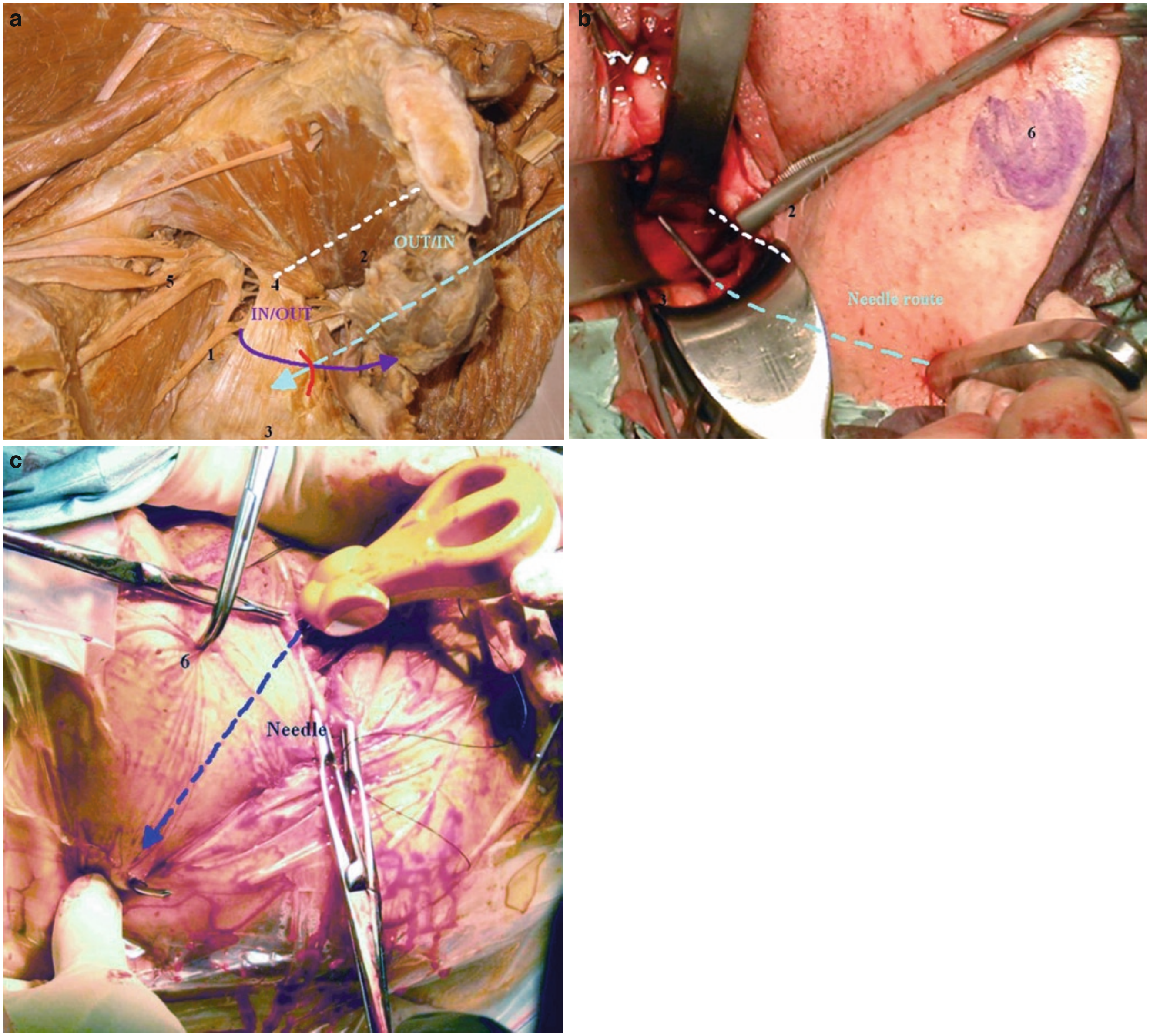

Fig. 11.5 The posterior arm: scrosciatic ligament. (**a**) Dissection. (**b**) Way OUT/IN Ischioanal route. (**c**) Way IN/OUT = transbuttock route "Short way." *1* Pudendal nerve, *2* Pudendal line, *3* Sacro-spinous ligament, *4* Sciatic spine, *5* Sciatic nerve, *6* Ischiatic tuberosity

At the end of surgery, the rectal examination allows to push laterally the posterior arms to make it supple. Too much tension of the posterior arms squeezes the rectum, which can be responsible for severe constipation. It is better to cut the outside end of the USS after the rectal relaxation of the posteriors arms, because these arms may go back inside the buttock for about 4–5 cm and may pass back through the sacro-spinous ligament; thus, the mesh would not hold enough. It is not necessary to close the small skin incisions (to introduce the arm), because the sutures are not comfortable for the patients.

We prefer the transpelvic arms than the fixation of the arms on the pelvic wall with hook, anchor, or glue. The transpelvic arms have two advantages:

1. The vaginal incision can be well closed outside of the patient before pulling the arms.
2. In the first week after the surgery, the tension-free sustentation of the arms permits the mobilization of the prosthesis when the patient stands up with a full bladder. We think that it may decrease the risk to have an overcorrection and avoids dysfunctions of the pelvic organs.

Experience and Discussion

Our experience is from three series of patients. Series 1 was between 1999 and 2002 and concerned 80 patients. It was the beginning of the experience. The prosthesis was 4 cm wide with only two ATO arms and one fixation on the sacrospinous ligament by nonresorbable suture. We had a lot of shrinking (12 cases) and lateral recurrences of the cystocele. We conclude that the prosthesis is not wide enough and other arms would be necessary to prevent lateral shrinking of the prosthesis (Tables 11.1–11.3).

Table 11.1 Arms complications

Series 2: – A.T.O = 346 : 0 complication – P.T.O (cross the pudendal line) = 346 5 Shrinking (dyspareunia)/2 erosion) 2 Pudendal pain
Series 3: – ATO = 164 : 0 complication – PTO (lateral to the pudendal line) = 164 0 Complication – USS = 166 : 1 Shrinking => dyspareunia

Table 11.2 Perioperative complications

	Series 2	Series 3
T° > 38°C	2%	2%
Mesh infection	2%	0%
Bladder injury	1.5%	0%
Pathological hematoma	3 %	0%

Series 2 no drain = 5 pathological hematoma
Series 3: drain = 0 pathological hematoma
(38 drains => 40–300 mL)

Table 11.3 Erosion and shrinking of the mesh

Series 1	Shrinking of the mesh	
	12 Patients (15%)	
Series 1	Shrinking of the mesh	
	Lateral cystocele : 12 patients (15%)	
	Series 2	*Series 3*
Exposition	3 (3%)	3 (3.5%)
Erosion	9 (4.5%)	0 (0%)
Pathological shrinking	6 (3%)	0 (0%)
Series 2	Shrinking of the mesh	
	Median high cystocele: 3 patients	

Series 2 was between 2002 and 2005 and concerned 205 patients. The prosthesis was with four arms and the mesh was sutured on the cervix by resorbable threads. The length of the mesh was 4–5 cm at the beginning of the experience and then 8 cm. The prosthesis was 6 cm wide. We observed disjunction between the cervix and the mesh, responsible for high cystocele recurrence. This is why we thought that the suture fixation is not strong enough and we propose the transuterosacral passage of the USS arms, to have a strong fixation between the mesh and the cervix. In this second series, the PTO directly crossed the Pudendal line and were very close the lateral vaginal sulcus. It is not always easy to prove Pudendal injury but we observed it in two cases. In any case of shrinking, the PTO is responsible for pain. This statement and a new anatomic study drove us to a new pelvic way for the PTO arm.

Series 3 was between 2005 and 2007 and concerned 94 patients. It was with the octopus prosthesis described above. We note that erosion is very rare. It is difficult to prove why. Perhaps, it is because the mesh is of better quality, but it seems that the more experience the surgeon has, the better the quality of the postsurgery result. The experience of TVM and Prolift seems to confirm that the improvement of the results depend on the experience of the surgeon.[10–12]

Conclusion

The technique to implant cystocele mesh with tension-free arms is now well known. The knowledge of the anatomy is fundamental for the surgeon:

- To know the landmarks to limit the risk of nerve, vessel, and visceral injuries during the implantation of the arms of the prosthesis.
- To have a good appreciation of the topography and good positioning of the prosthesis at the end of surgery.

The anterior and posterior arms are really the support of the prosthesis. We hope that the lateral arms (PTO) just limit the lateral shrinking of the mesh. The arms must be stretched without tension, because they go through muscle only and tension would injure the Obturator and Levator muscles. It might result in bad positioning of the arms, too close to the vaginal sulcus, increasing the risk of mesh shrinking and of vaginal erosion.

It is too soon to estimate the results of this surgery compared to free mesh with no arms, facial surgery, and laparoscopic surgery. But the first results prove the feasibility and the low morbidity of this technique.

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Coexisting Cystocele and Stress Urinary Incontinence: Sequential or Concomitant Surgical Approach?

12

Roger Lefevre and G. Willy Davila

Background

One of the significant attributes of pelvic reconstructive surgery is the availability of techniques with fairly immediate and demonstrable effects to the patient. Women seeking surgical help for an uncomfortable vaginal bulge can reasonably expect resolution of their problem upon discharge from the hospital. The same cannot always be said when a patient has coexistent stress urinary incontinence or voiding dysfunction. A well-known frustrating situation for both the surgeon and his/her patient is the woman who returns home after reconstructive surgery only to deal with de novo, persistent, and even sometimes worsened urinary problems. Although, these symptoms are usually temporary, even the previously well-counseled patient can still have a difficult time accepting such persistence of symptoms.[1]

The coexistence of anterior vaginal prolapse and stress urinary incontinence is a common challenge for the managing reconstructive surgeon. Bai et al. reported a 63% rate of coexisting pelvic organ prolapse and stress urinary incontinence.[2] In the past, surgeons have attempted to confront this dilemma by managing the two conditions with a single procedure. The suburethral "Kelly plication" and the "Bologna" procedure are examples of procedures that yielded less than acceptable outcomes.[3]

Unfortunately, repairing significant prolapse may not restore continence predictably, even if a dedicated sling procedure is performed. Patients with symptomatic stress incontinence may readily discuss treatment options. However, those with significant prolapse who are continent represent a special group of patients who require special attention and counseling. In this chapter, we will review the evaluation and management of women with significant anterior vaginal wall prolapse and stress incontinence, be it symptomatic or not.

Evaluation

A question that still yields lengthy discussions among pelvic surgeons is whether or not incontinence should be addressed based only on a patient's reported symptoms or whether it should be actively ruled out during preoperative evaluation? Some patients with vaginal wall prolapse will have their incontinence "unmasked" during their pelvic examination when the bulge is reduced or during preoperative urodynamics testing. The incidence of what has been termed "occult incontinence" has been observed to increase with worsening (grade 3–4) prolapse.[4] Depending on the reduction tool used, 36–80% of patients will demonstrate stress urinary incontinence during their preoperative evaluation. External compression of the urethra from an enlarging posterior wall prolapse and urethral kinking[5] remain the most notable theories offered to explain this phenomenon.

A major criticism of preoperative "occult incontinence" identification efforts has revolved around the lack of standardization of the tools and techniques used. Patient discomfort and risk of urethral occlusion represent some of the various factors that can influence the choice of a prolapse reduction device utilized. Various studies have compared tools utilized for prolapse reduction during urodynamic testing but the data remains unclear. Our group reported on 36 patients with advanced vaginal prolapse randomized to using the lower blade of a plastic speculum, a ring pessary, or a vaginal packing versus not reducing the prolapse at the time of preoperative urodynamics evaluation. All three devices statistically increased the detection rate of occult incontinence compared to the unreduced group but showed no significant difference when compared to each other.[6] Based on our findings, we currently use the lower blade of a plastic speculum in patients with advanced vaginal prolapse.

The preoperative counseling session in these cases can often be quite challenging when trying to explain to the self-perceived "continent" patient that she may need an anti-incontinence procedure in addition to her prolapse repair. This discussion is crucial, as the occurrence of de novo urinary incontinence is commonly perceived as a failure of the primary operation by the patient.

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Liang et al. demonstrated that at the time of vaginal hysterectomy, performing a TVT sling procedure on self-perceived "continent" patients with a positive prolapse reduction test can significantly prevent the development of postoperative SUI.[7] This study differentiates itself from similar trials because it reported a cure rate of 90.6% for those patients treated for their occult incontinence as well as a 64.7% rate of postoperative SUI in those that did not. All patients with negative prolapse reduction testing remained dry postoperatively.

The Colpopexy and Urinary Reduction Efforts (CARE) trial demonstrated a clear benefit to continent women undergoing abdominal sacrocolpopexy for vaginal vault prolapse who had a concomitant prophylactic Burch colposuspension causing the early cessation of enrollment after their first scheduled interim analysis.[8] Interestingly, 56% of patients who randomized to the nonintervention group remained continent postoperatively. This can be interpreted as performing an unnecessary intervention in more than half of the patients and reaffirms the need for being more selective when deciding on who should undergo an anti-incontinence procedure.

To that effect, Visco et al. published a sub-analysis of the (CARE) trial by extracting the women with occult incontinence unmasked at their preoperative urodynamics evaluation and reporting on their corresponding surgical outcomes with and without undergoing an anti-incontinence procedure.[9] First and foremost, this study confirmed the benefit of prolapse reduction during the evaluation of patients with vaginal wall prolapse. This method has long been a common practice amongst urogynecologists and has been criticized extensively in the literature for lack of data. Amongst all the continent women, SUI was noted in 27% when their prolapse was reduced as compared to only 3.7% of women who were not. Postoperatively, the patients who randomized to the "no Burch" group were analyzed retrospectively as to whether occult incontinence was unmasked or not. 58% of those with positive testing went on to have postoperative SUI as opposed to 38% of women with negative prolapse reduction testing. Overall, although the results of the two prior studies pertain specifically to an abdominal approach to prolapse and stress incontinence, they can guide vaginal surgeons to fine tune their surgical management algorithm.

The "Outcomes following vaginal Prolapse repair and mid-Urethral Sling" (OPUS) trial is currently enrolling patients and aims to provide similar answers from the vaginal approach to prolapse and incontinence.[10]

The role of preoperative urodynamics is particularly important in the patient with significant vaginal prolapse and cannot be overemphasized.

Management Options

The dilemma arises in choosing a sequential surgical approach versus performing a combined repair of the patient's cystocele and stress urinary incontinence. In the *sequential* approach, the cystocele is addressed primarily and the patient can be followed postoperatively for persistence or resolution of her incontinence. An outpatient sling procedure can be performed later as indicated. In the *concomitant* surgical approach, both problems are addressed during one operation. The former surgical management plan can seem intuitive especially when one considers the impact that the anti-incontinence procedure may have on the prolapse repair and vice versa.

Indeed, studies have identified anti-incontinence procedures as a risk factor for cystocele recurrence. In 1996, Kohli et al. reported on a retrospective analysis of patients with anterior wall prolapse after having an anterior colporrhaphy with or without needle suspension.[11] There was a clear difference in cystocele recurrence rates after 13 months with 33% in the colporrhaphy/suspension group versus 7% in the colporrhaphy only patients. This discrepancy was theorized to be secondary to the surgical dissection required for the needle suspension and possible iatrogenic creation of paravaginal defects. Additionally, other procedures such as urethropexy[12] and sacrospinous ligament suspension[13] have been shown in the literature to increase the risk of recurrence in anterior wall prolapse.

Some pelvic surgeons elect to perform both the cystocele and anti-incontinence procedures simultaneously. The increasing popularity of suburethral slings has provided enough data to independently report on their effect. Goldberg et al. reported a sub-analysis of their group's original study on the effect of polyglactin 910 mesh on the recurrence of anterior and posterior wall prolapse.[14] From that population, they isolated those who had undergone suburethral sling placement to Cooper's ligament as part of their pelvic reconstruction and determined its independent effect on cystocele recurrence. The sling group only had a 19% cystocele recurrence rate at 1-year follow-up as compared to 42% in the non-sling group. Prior to that study, Cross et al. had reported on 42 women with coexisting anterior wall prolapse and SUI. They also found a protective association between pubovaginal slings and a lower cystocele recurrence rate of 8.3% at 20 months.[15]

While the sequential approach to coexisting prolapse and stress incontinence allows for more precise counseling and avoids imposing potential negative changes in urinary function on a patient, it is often criticized from the financial perspective. In countries with non-socialized health care system, some surgeons have been known to wait a minimum of 12 weeks after the onset of postoperative stress incontinence before intervening surgically. This delay may correspond to the "global period" mandated by insurance companies in the USA and can add a negative "financial gain" connotation to the surgeon.

Additional factors have to be considered when deciding on a *sequential* versus *concomitant* approach to prolapse and incontinence. From a patient's point of view, having both problems addressed within one admission can have significant health and social benefits, not to mention cost. Although significant improvements in technology have developed over the past decades, the concern regarding being subjected to anesthesia repeatedly remains a valid one. One operation also translates into one recovery period and less time lost from one's personal occupation and physical activity/exercise routine. As surgeons, we often focus on minimizing surgical failures and occasionally lose sight of the functional and social consequences of our recommended management options to our patients. Nowadays, more women are primary financial providers for their families and can be significantly affected by the loss of wages resulting from multiple convalescence periods. Unfortunately, there is literature to support both concomitant and sequential prolapse/SUI management strategies, and the dilemma regarding which to adopt persists.

Adverse Effects: Voiding Dysfunction

Identification

De novo postoperative voiding dysfunction can have a devastating impact on a patient with coexisting prolapse and SUI. It is characterized mainly by the development of symptoms of overactive bladder (urgency and frequency), obstructive voiding patterns, and urinary retention. It is described objectively based on pressure-flow studies during urodynamics, post-void residual measurements, X-ray appearance of the bladder neck, or a combination of the aforementioned. Chassagne et al. prospectively followed 35 patients who were clinically obstructed and compared urodynamics parameters to 124 controls. Using a combination of $Q_{\max} \leq 15$ mL/s and $P_{\mathrm{det}} > 20$ cm of H_2O, they were able to refine the diagnosis of bladder outlet obstruction (BOO) with 91.1% specificity in women.[16] Nitti et al. retrospectively reviewed 331 charts of patients having undergone urodynamics testing for voiding dysfunction. They identified 76 patients with evidence of obstruction by fluoroscopy and demonstrated the benefits of adding video urodynamics to pressure-flow studies in order to improve the diagnosis of BOO.[17]

The risk of postoperative urinary retention, especially after performing prolapse and anti-incontinence repairs concomitantly can be a crucial piece of information during preoperative counseling. Sokol et al. retrospectively reviewed 266 patients and found similar rates of retention (11.2 vs 11.3%) and mean days to void (8 vs 5) after TVT with or without prolapse surgery.[18] Bhatia and Bergman studied the risk of retention after Burch colposuspension and found a 12-fold increase in patients with a detrusor pressure of <15 cm of H_2O on preoperative evaluation.[19] Similarly, regarding suburethral slings, Hong et al. reported a 27.3% rate of urinary retention amongst TVT patients who demonstrated a peak flow <15 mL/s on preoperative pressure flow studies.[20]

Data on the predictive value of preoperative urodynamics evaluation are variable as well. The aforementioned rates of postoperative voiding dysfunction associated with slings have to be considered when counseling a self-perceived continent patient about their need of an anti-incontinence procedure. De Tayrac et al. followed 48 women with coexisting prolapse and SUI over a 20-month period after prolapse repair alone versus performing a concomitant TVT procedure. They found a 27.3% incidence of postoperative dysfunction in patients with occult incontinence demonstrated during preoperative urodynamics compared to 13.3% in those with overt SUI[21]. Our group reported a 26% postoperative voiding dysfunction rate found at 3 months among 59 women who underwent a TVT with or without prolapse repair.[22] An abnormal preoperative uroflow pattern/configuration, a peak flow <15 mL/s or a concomitant vault suspension procedure was highly predictive of postoperative voiding dysfunction.

The definitions of urinary retention and voiding dysfunction between reported studies differ as much as the management style amongst urogynecologists when it comes to concomitant versus sequential approaches. The most noticeable consistency here is the variability of the reports of voiding dysfunction after anti-incontinence procedures. It illustrates the difficulty faced by pelvic surgeons in interpreting the available literature and subsequently making surgical decisions.

Management

Most commonly, following the resolution of postoperative edema and pain, most patients return to their usual voiding routine but some still require temporary bladder drainage or intermittent self-catheterization (ISC). The timing of intervention in patients with symptoms of postoperative voiding dysfunction represents yet again another challenge for the pelvic surgeon.

Conservative modalities such as ISC and pharmacotherapy can be attempted first and help buy time during the postoperative period. They are often effective and should be attempted first when such complications arise after an anti-incontinence procedure. As mentioned above, in the USA, some surgeons try conservative measures for about 12 weeks before surgically intervening. This is considered for the most part arbitrary, and authors such as Rosenblum have found that symptoms of postoperative voiding dysfunction persisting beyond 4 weeks are unlikely to resolve without intervention.[23]

Pharmacological agents such as urecholine, baclofen, and alpha blockers are some of the most commonly attempted during the early postoperative period in patients with dysfunctional voiding. Preoperative pressure-flow study and urethral electromyography (EMG) should be reviewed as they can prove to be useful in the selection among the above agents. In patients who demonstrated non-relaxation of the urethral sphincter with increased recruitment of pelvic floor musculature (absent silencing or increased EMG activity) during voiding, a presynaptic Gamma Amino Butyric Acid (GABA-B) agonist such as baclofen or alpha blocker such as prazosin can help improve flow. In patients with weak preoperative detrusor pressure who are experiencing postoperative obstructive symptoms, urecholine can be attempted instead. As a cholinergic agent, it helps activate the parasympathetic nervous system and can significantly increase the tone of the detrusor muscle to help initiate micturition, although contraction pressures themselves do not increase.

Pharmacotherapy can also be used in conjunction with ISC to help with bladder retraining. In motivated and physically able patients, when the post-void residual is consistently greater than 100 cc, intermittent self-catheterization has been shown to be a useful adjunct. Once the patient demonstrates understanding and proficiency in performing this procedure, we typically recommend ISC two to three times daily, especially upon awakening.

Unfortunately, conservative measures can fall short in providing complete resolution of obstructive voiding symptoms after an anti-incontinence procedure. Postoperative multichannel urodynamics are often necessary to elucidate the nature of the voiding dysfunction and can provide objective evidence of obstruction. Transvaginal urethrolysis with sling transection has shown low morbidity and great efficacy while maintaining acceptable continence rates[24.] At our center, we have made similar observations even after cases of sling transection. 12–16 weeks is the required time for the entire polypropylene sling to incorporate itself into the periurethral tissues. Once the obstruction is objectively confirmed, we tend to proceed with the transection of the sling at the mid-suburethral segment. It is not necessary to extract the arms as their role in providing continued periurethral support can help maintain continence (Fig. 12.1).

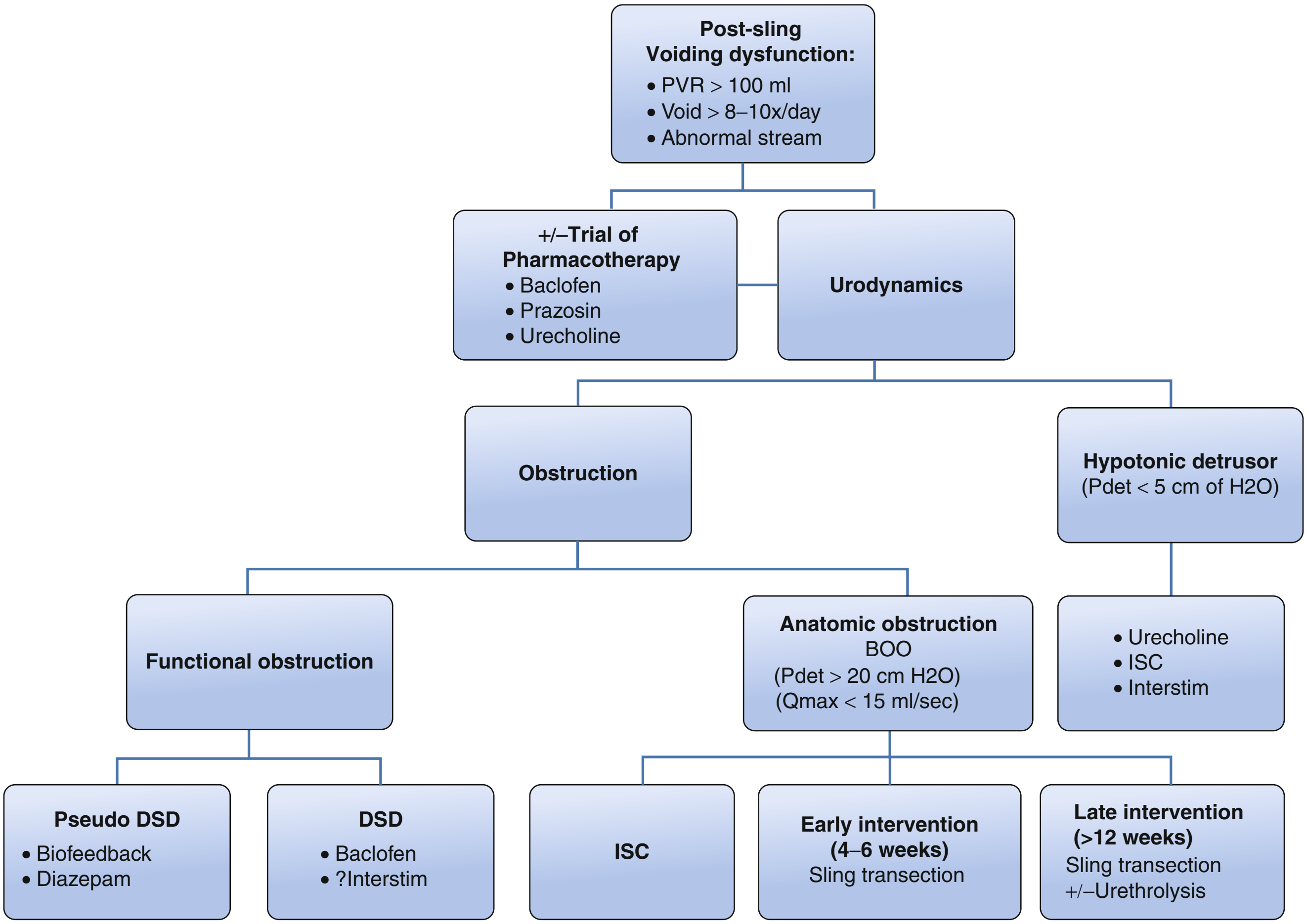
Post-sling
Voiding dysfunction:
• PVR > 100 ml
• Void > 8–10x/day
• Abnormal stream
+/–Trial of Pharmacotherapy
• Baclofen
• Prazosin
• Urecholine
Urodynamics
Obstruction
Hypotonic detrusor
(Pdet < 5 cm of H2O)
Functional obstruction
Anatomic obstruction
BOO
(Pdet > 20 cm H2O)
(Qmax < 15 ml/sec)
• Urecholine
• ISC
• Interstim
Pseudo DSD
• Biofeedback
• Diazepam
DSD
• Baclofen
• ?Interstim
ISC
Early intervention
(4–6 weeks)
Sling transection
Late intervention
(>12 weeks)
Sling transection
+/–Urethrolysis

Fig. 12.1 Algorithm for management of post-sling voiding dysfunction

Minimizing the development of potential symptoms associated with obstructive suburethral sling placement such as de novo voiding dysfunction and irritative voiding symptoms (OAB-type) is crucial. Even the well-counseled patient may view the development of such symptoms as a complication. In this regard, a sequential approach may lead to an overall more satisfied patient in the long run. Expanded data is clearly needed to be able to make more conclusive recommendations.

Unique Circumstances

No Hypermobility

A cystocele is believed, for the most part, to develop as an anterior vaginal wall fascial tear from the vaginal apex or peri-cervical ring that progressively enlarges. The patient who retains adequate periurethral support can in fact present with a cystocele and stress incontinence *without* any urethral hypermobility. In this situation, the cystocele should be repaired with care taken to securely reattach the endopelvic fascia to the vaginal apex or cervix as this consists in the original central point of weakness. The dilemma lies in whether or not to address any coexistent incontinence. One could argue that a suburethral sling would not be of much use in this situation due to the lack of urethral hypermobility. A thorough discussion should be held between the patient and her surgeon favoring a sequential surgical approach. The patient should be counseled regarding transurethral bulking agent injections or pelvic floor rehabilitative therapy, both of which can be successful when there is no urethral hypermobility.

Intrinsic Sphincter Deficiency

Intrinsic sphincter deficiency (ISD) is another topic of much debate in the field of urogynecology and its management is very variable amongst pelvic surgeons. Whether defined as a maximal urethral closure pressure of <20 cm of H_2O or a Valsalva leak point pressure of <60 cm of H_2O, most would agree that a pubovaginal/retropubic sling is the recommended option in patients presenting with ISD with urethral hypermobility and urethral bulking agent injections for those without. The differences in opinions are centered mostly on issues such as the location of the sling (bladder neck or mid-urethra), its mechanical properties (elasticity, tensioning ability), and the choice of material (synthetic vs cadaveric vs fascia lata).

TVT is one of the most popular suburethral sling performed for stress incontinence worldwide. However, the data regarding its use in patients with ISD is limited. There are only a handful of published case series and no randomized trials comparing retropubic slings have been reported (Table 12.1).

Traditionally, when treating ISD, bladder neck retropubic slings have been employed. Only recently has this condition been approached with mid-urethral tapes. Pelvic surgeons continue to debate regarding the mechanical properties and tensioning abilities of their preferred slings. Our group recently reported our experience with a retrospective study comparing the use of an elastic mid-urethral sling (TVT) to a nonelastic bladder neck sling (I-STOP) for patients with ISD with hypermobility. There was no difference in success rates in any of the outcomes measured. De novo urge incontinence rates were similar in I-STOP and TVT groups (13.3% vs 11.6%, respectively, $p = 1.00$). The resolution of urgency symptoms postoperatively was greater in the I-STOP group (67.5% preoperatively to 30%, $p < 0.001$) versus the TVT patients (35–20%, $p = 0.082$).[29] With these supportive facts, we elect to perform a retropubic, nonelastic, bladder-neck sling at the time of a cystocele repair in patients with ISD.

The choice of a “sequential” versus “concomitant” management strategy can be controversial for patients presenting with both anterior wall prolapse and ISD with hypermobility. They should be counseled extensively and made aware of the greater likelihood of persistent postoperative incontinence if a sling is not placed at the time of the repair. This conversation becomes even more crucial when dealing with a woman with “occult” ISD unmasked during preoperative urodynamics testing. Managing the two conditions concomitantly may yield a higher satisfaction rate for both patients and surgeons.

Table 12.1 TVT in ISD patients

Study	ISD parameters used	(*N*)	Follow-up (months)	Cure rate (%)
Ghezzi et al.[25]	MUCP <20 cmH2O and VLPP <60 cmH2O	35	12.5 (3–36)	91
Rezapour et al.[26]	MUCP <20 cmH2O	49	48 (36–60)	86
Paick et al.[27]	VLPP <60 cmH2O	61	10.5 (6–52)	82
Lapis et al.[28]	MUCP <20 cmH2O	37	26 (22–30)	82.2

Bladder atony or even a weak detrusor pressure ($P_{det} < 5$ cm H_2O) during preoperative pressure flow study often causes surgeons to be hesitant to perform a sling at the time of a cystocele repair. They may elect for the "sequential" approach and plan for a subsequent in-office bulking agent injection or outpatient sling procedure. This strategy will likely reduce the risk of postoperative voiding dysfunction or obstructive symptoms but at the same time may yield lower continence rates among these women with ISD.

The placement of a 14 Gauge suprapubic catheter (Bonanno BD NJ, USA), when performing concomitant procedures, has worked well for our group in the management of these patients. It allows for drainage of residual urine during postoperative bladder retraining. Most of our patients are able to have it removed with normal post-void residuals within 7–10 days.

Mixed Incontinence

When dealing with mixed incontinence (MI), preoperative urodynamic testing proves its utility in providing the surgeon with the ability to counsel women regarding the rates of improvement or resolution of their urge incontinence symptoms based on the chosen intervention. It can also provide some guidance on whether or not to adopt a sequential surgical approach.

In general, it is essential to correlate the findings of any evaluation modality to the patient's presenting complaint. For example, when the diagnosis of "mixed incontinence with urge > stress" is obtained, one may indeed consider starting an anticholinergic medication first and delaying the performance of the cystocele repair. If the presenting symptoms improve, this may allow the surgeon to hold off on any anti-incontinence procedure and avoid potential complications such as de novo obstructive symptoms. When dealing with "stress > urge MI," the patient who undergoes a sling can expect about a 50% improvement in her urge incontinence symptoms in addition to the expected stress incontinence success rate related to the particular suburethral sling used during the surgery.

Some patients have their quality of life equally affected by both symptoms or cannot truly provide any insight into any predominance from stress or urge incontinence. Unfortunately, in this case, the current available literature does not provide any guidance into which component to address first. It is the author's opinion that because urge incontinence is often accompanied by a sense of "loss of control," its resolution would provide the greatest impact to the patient. Hence, when dealing with a patient with mixed incontinence (stress = urge), we tend to offer medical management whether or not the patient elects to undergo an anti-incontinence sling procedure.

Preoperative Urinary Retention on UDS

Due to Prolapse

As previously mentioned, the concept of urethral "kinking" with advanced anterior wall prolapse can be a significant pseudo-continence mechanism. As such, a patient's bladder may have to generate a stronger contraction to overcome this anatomic outlet obstruction in order to urinate. The clinical finding of an elevated post-void residual should not automatically generate hesitations about addressing a patient's stress incontinence. A reassuring indication at the time of preoperative multichannel urodynamics testing is the ability of a patient with urinary retention to completely void the entire amount infused during a cystometrogram once the cystocele is reduced. In this case, the benefit of addressing her stress incontinence with a suburethral sling outweighs the risk of voiding dysfunction.

Persistent Urinary Retention Despite Reduction

The situation is quite different when dealing with a patient with incomplete emptying or persistent urinary retention despite prolapse reduction. Prior to considering any anti-incontinence procedure, the cystometrogram should be reviewed for any evidence of decreased compliance or overactivity. If present, a voiding cystogram should be ordered to rule out any vesicoureteral reflux as this would represent an absolute contraindication for a surgeon to address the symptoms of stress incontinence with a sling.

As mentioned above in the section for ISD patients, an accurate pressure-flow study is crucial and can provide critical information to help guide a surgeon's clinical decision. Some patients with a neurological lesion will exhibit a non-relaxing urethral sphincter along with a concomitant increased recruitment of their pelvic floor muscles during micturition. They carry the urodynamic diagnosis of detrusor sphincter dysynergia (DSD) and can demonstrate an incomplete/abnormal void. Postoperatively, they usually benefit from early initiation of pharmacologic agents such as Baclofen or Prazosin. On the other hand, some patients have "learned" behaviors resulting in a voiding mechanism that mimics that of DSD patients but have no true neurologic lesions. These women with "pseudo-DSD" can require a combination of muscles relaxants (Diazepam) and/or pelvic floor retraining in order to alleviate their urinary retention.

Other patients with weak detrusor contractility during a pressure-flow study should also be considered carefully and not automatically deter the surgeon from performing a sling. Pharmacotherapy and placement of a suprapubic catheter at the time of surgery can help for postoperative bladder retraining.

Conclusion

We propose the following algorithm (Fig. 12.2) as a guide to managing patients with advanced vaginal wall prolapse with coexistent SUI. The authors' views on the combined surgical approach are noted in Table 12.2.

"I will prescribe regimens for the good of my patients according to my ability and my judgment and never do harm to anyone" is a critical part of the Hippocratic Oath that we take as physicians. Considering the frequency with which female pelvic organ prolapse is accompanied with stress urinary incontinence, it is critical for the surgeon to be conscientious of the postsurgical and social implications of adopting a combined or sequential approach. The treating physician has to develop his/her own clinically and ethically responsible algorithm for women suffering simultaneously with anterior wall prolapse and incontinence. With the introduction of surgical kits and the increasingly favorable data available regarding suburethral sling use, one can see why the combined approach is becoming more popular among pelvic surgeons. The lesser exposure to anesthesia, the proposed additional benefit of reduction in cystocele recurrence and the nullification of surgeon's potential financial motivation all favor treating prolapse and incontinence concomitantly. With careful and considerate preoperative counseling, both the surgeon and his/her patient can work together in managing the recognized postoperative incidence of voiding dysfunction that accompanies anti-incontinence procedures.

Table 12.2 Pro(s) versus con(s) for combined surgeries

Combined approach	
Pro(s)	Con(s)
Protective for cystocele recurrence [14,15] (suburethral slings)	Increase postoperative voiding dysfunction [21]
Prevents de novo SUI [8]	Unnecessary treatment [9]
Decrease costs on health care system	Comprehensive counseling
Single convalescence period	

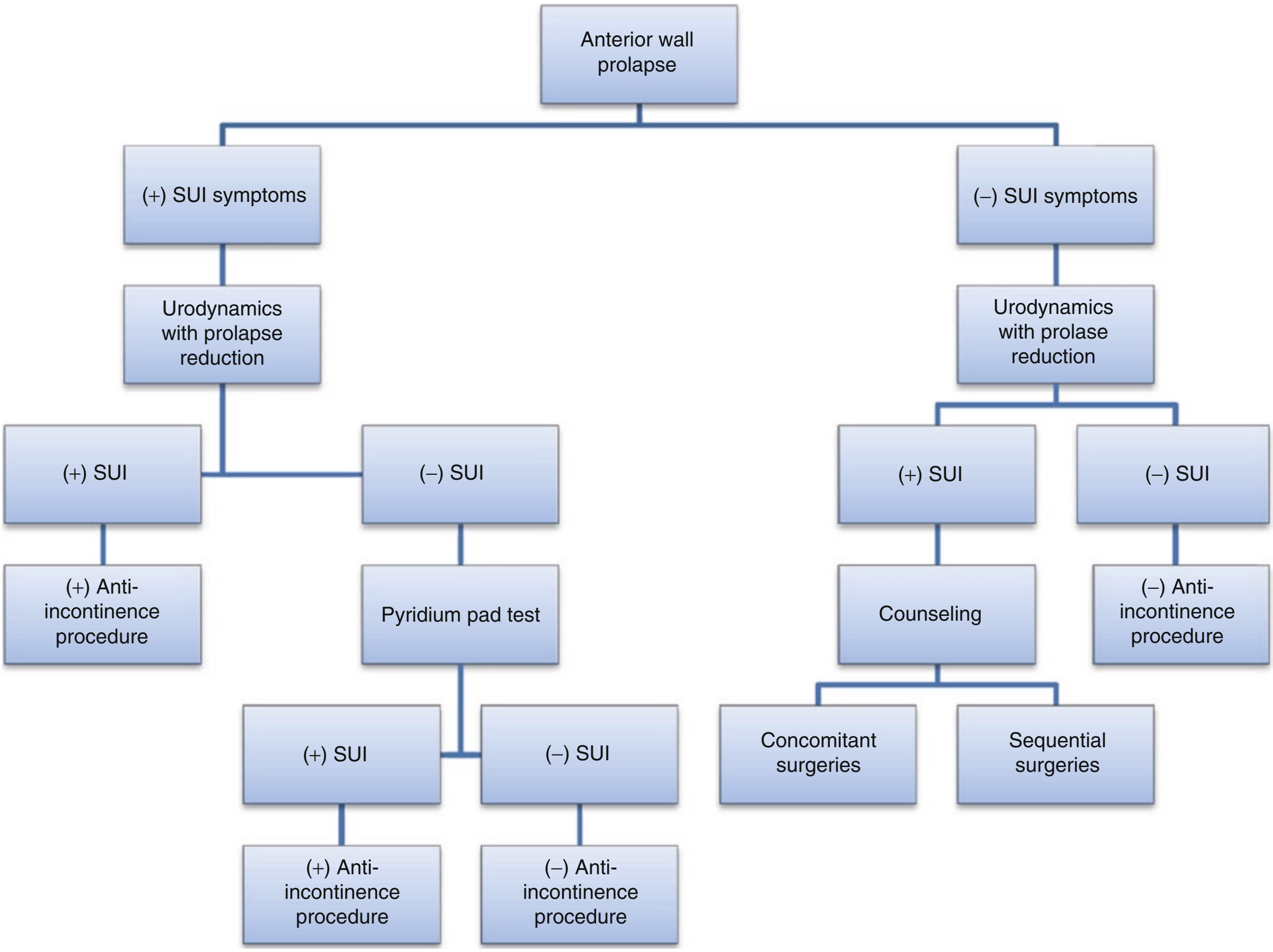
Anterior wall prolapse
(+) SUI symptoms
(–) SUI symptoms
Urodynamics with prolapse reduction
Urodynamics with prolase reduction
(+) SUI
(–) SUI
(+) SUI
(–) SUI
(+) Anti-incontinence procedure
Pyridium pad test
Counseling
(–) Anti-incontinence procedure
(+) SUI
(–) SUI
Concomitant surgeries
Sequential surgeries
(+) Anti-incontinence procedure
(–) Anti-incontinence procedure

Fig. 12.2 Algorithm for management of concomitant cystocele and SUI

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Simultaneous Repair of Stress Urinary Incontinence (SUI) with the Cystocele Mesh

13

Peter von Theobald

Definitions and Diagnosis

Dealing with stress urinary incontinence (SUI) associated with pelvic organ prolapse (POP) is a frequent but controversial matter. Not only is surgical strategy debatable, but also the definition and the diagnosis of SUI. There is general agreement about so-called overt SUI (patient with POP complaining of urinary leakage at stress), but no clear definition of "occult SUI" exists in literature. According to Haessler[1], occult SUI exists when "…*leaking occurs with Valsalva manoeuvers after reduction of the prolapsed*," and concerns 36–80% of POP patients. That is, after POP surgery, a patient without preoperative SUI may experience occult SUI if no specific treatment is performed. According to Visco[2], however, prolapse reduction to predict the risk of SUI is not evidence-based; it is not even standardized. Furthermore, it is unclear within the literature whether "occult" is synonymous with "potential," "masked," "latent," "hidden," or "iatrogenic."

It is also unclear whether "de novo" has yet a different meaning. According to Kleeman[3], "de novo" SUI occurs in 1.9% of POP patients if one considers patients without preoperative "occult" SUI (at prolapse reduction test). But in some trials, like the CARE study comparing sacrocolpopexy with and without colposuspension[4,5], "de novo" SUI refers to patients without "overt" preoperative SUI becoming incontinent after the procedure. Those studies report a 45.2% rate of "de novo" SUI in the control group without colposuspension. The same type of trial published by Constantini[6], in which "de novo" was defined as postoperative SUI after negative preoperative POP reduction test, shows a rate of 3.1% of "de novo" SUI in the control group without colposuspension. These results are obviously unreliable because each publication uses a different definition of "de novo." For the patient with POP, who was not leaking before operation, experiencing postoperative SUI will be considered as "de novo." The patient's interest is in whether SUI can be predicted or prevented, but explaining that the symptoms do not constitute "de novo" but "occult," "hidden," or "potential" SUI will make no sense to the patient. Clear definitions should be given by international continence societies for all situations other than "overt" SUI: positive and negative preoperative cough test with prolapse reduction and SUI following both clinical findings. Despite discrepancies among definitions, the literature consistently show that postoperative SUI is significantly more likely to happen in patients with positive preoperative tests[4–6], meaning that these patients have an anatomical defect that requires a treatment.

Treatment of Concomitant SUI

Another debate exists about whether overt (and "occult") SUI should be treated at the same time as the POP or at a second operation some months later. No comparative trials have been reported in the literature to support this latter strategy concerning results. The only argument for such a strategy might be financial, depending on the health system. In many countries, performing two different operations is better paid and makes it possible to be reimbursed for the meshes through health insurance. Obviously, a single operation is preferred by the patient. To perform both repairs at the same time, there are two solutions: insert a suburethral sling through a second incision on the anterior vaginal wall or use a cystocele mesh whose anterior arms are inserted as a suburethral sling through a single incision (Figs. 13.1–13.3). Several publications are available concerning vaginal prolapse repair with mesh and concomitant SUI repair.

SUI Repair with an Additional Tape

De Tayrac[7] has published a retrospective case control series of 29 patients with overt SUI and 19 patients with occult SUI, all presenting a cystocele requiring a vesico vaginal

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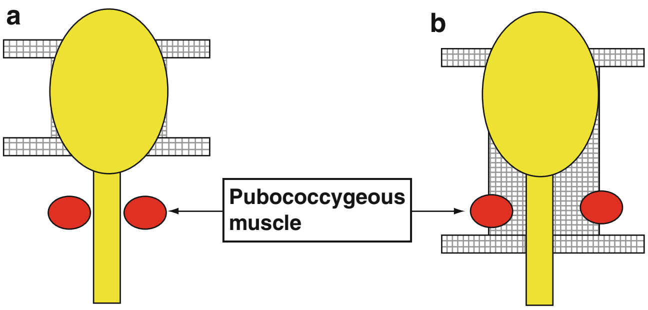

Fig. 13.1 (**a**) The four armed transobturator mesh for cystocele. (**b**) The four armed mesh for cystocele with concomitant

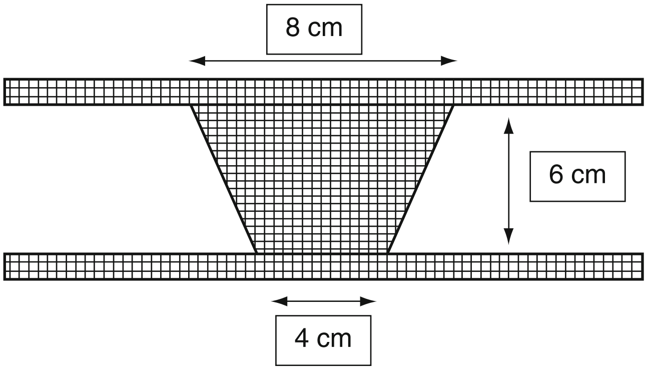

Fig. 13.3 Dimensions of the mesh

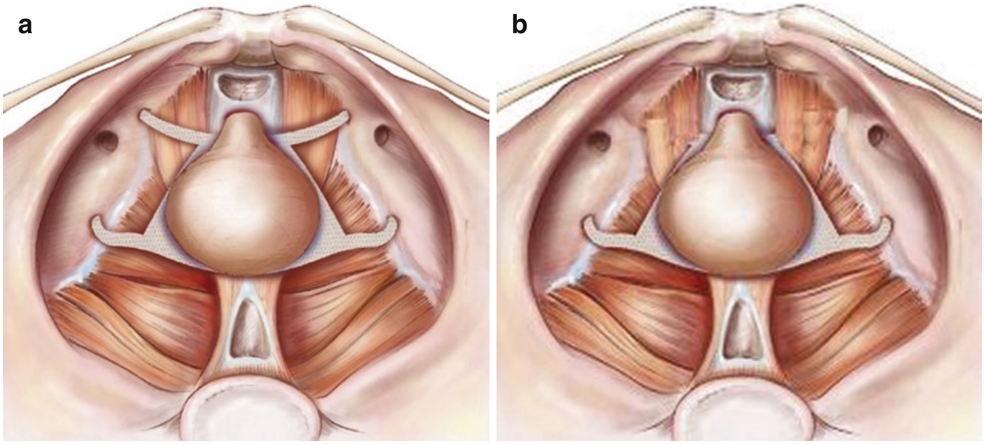

Fig. 13.2 (**a**) The four armed mesh for cystocele from above. (**b**) The four armed mesh for cystocele and SUI from above

mesh repair. SUI was treated by an additional retropubic TVT in 15 of the overt SUI patients and in 11 of the occult SUI ones. Postoperative SUI rates after 2 years were 6.7% versus 35.7% in the overt SUI group (results were significant) and 0% versus 12.5% in the occult SUI group (results were nonsignificant). But voiding dysfunction occurred in the overt SUI patients in 13.3% of the TVT group versus 0% in the control group ($p > 0.05$) and in the occult SUI patients in 27.3% of the TVT group versus 0% in the control group ($p < 0.05$). He concludes that in patients with preoperative SUI, TVT is more efficient than prosthetic cystocele repair alone to prevent postoperative SUI, without differences in voiding dysfunction and in patients with preoperative occult SUI, prosthetic cystocele repair is as efficient as TVT, with a decreased risk of voiding dysfunction. The problem here is that the series is very small and retrospective. The author, however, seems to show that an anterior cystocele mesh may be somehow effective for SUI repair in occult SUI patients with POP. These results are similar to the findings of the Constantini series with sacrocolpopexy associated or not to colposuspension.[6] This is contradictory to the randomized trial of Hiltunen[8] comparing anterior colporraphy to fascia plicature reinforced with mesh in 201 patients with 1-year follow-up. Twenty-three women (23%) with mesh and 9 (10%) with no mesh reported stress urinary incontinence ($p = 0.02$). Hiltunen concluded that anterior colporrhaphy, reinforced with tailored mesh significantly reduced the rate of recurrence of anterior vaginal wall prolapse compared with the traditional operation. However, it was associated more often with stress urinary incontinence.

Meschia[9] compared TVT to suburethral plicature in 50 patients with occult SUI associated to POP and found significant decrease in postoperative SUI rates in the TVT group (8% vs 44%). But in the TVT group, de novo urge incontinence was 12% versus 4% in the other group. Two points must be discussed here. First, the high postoperative SUI rate in the control group of these occult SUI patients was 44%. This result was very different from other series (between 1.9% and 23%).[3,6–8] Second, the cystocele repairs have been performed without mesh. Thus, the therapeutic effect may be different.

SUI Repair with the Same Tape as Cystocele

Sergent[10,11] has published a prospective series of 103 patients with cystocele and SUI; after a follow up of 32 months, 69% were dry, 20% improved, and 11% failed. Our personal series is a retrospective case control study comparing 60 patients between 2003 and 2005 with SUI alone treated by transobturator tape (IVS 04, TYCO, polypropylene multifilament) to 60 patients with cystocele and overt or occult SUI (self-tailored four-armed prosthesis of polypropylene multifilament, TYCO). Postoperative failure rate for SUI is 15% in patients with SUI alone and 10% in patients with cystocele and SUI. (Table 13.1) De novo urge incontinence was 10% and 11% in the two groups. This means that the vaginal dissection between level 2 and level 3, under the trigone, crossing the pubococcygeus levator muscle plane and putting a mesh in this layer, does not interfere with bladder stability. The only postoperative significant difference was the incomplete voiding rate defined as residuals superior to 100 mL, 48 h after procedure. This series of ours was a preliminary series. The erosion rate was 7% after 3 years of follow-up. A prospective trial with light monofilament meshes (Quadra, Covidien) is currently running.

The operative technique is shown in Figs. 13.4–13.9. A midline full thickness incision is performed on the anterior vagina extending up to 1 cm from the urethral meatus. The bladder is dissected away from the vaginal wall, leaving the Halban's fascia on the epithelium. The paravesical fossas are opened until the ischial spine and the arcus tendineous of the levator ani are reached. The paraurethral spaces are opened up to the ischiopubic rami. Between the paravesical fossas (level 2) and the paraurethral spaces (level 1), the internal part of the levator ani plate, the pubococcygeus muscle, is visible and very adhesive to the proximal urethra and the vaginal wall. Its thickness varies from one patient to another but usually, it is about

Table 13.1 Results of the personal case control series

		SUI	SUI + cystocele
Preoperative	Urge	37%	11%
	Voiding dysfunction	0.3%	8%
Postoperative	SUI	15%	10%[a]
	Urge	25%	17%[a]
	De novo urge	11%	10%[a]
	Voiding dysfunction	10%	4%

[a] n.s.

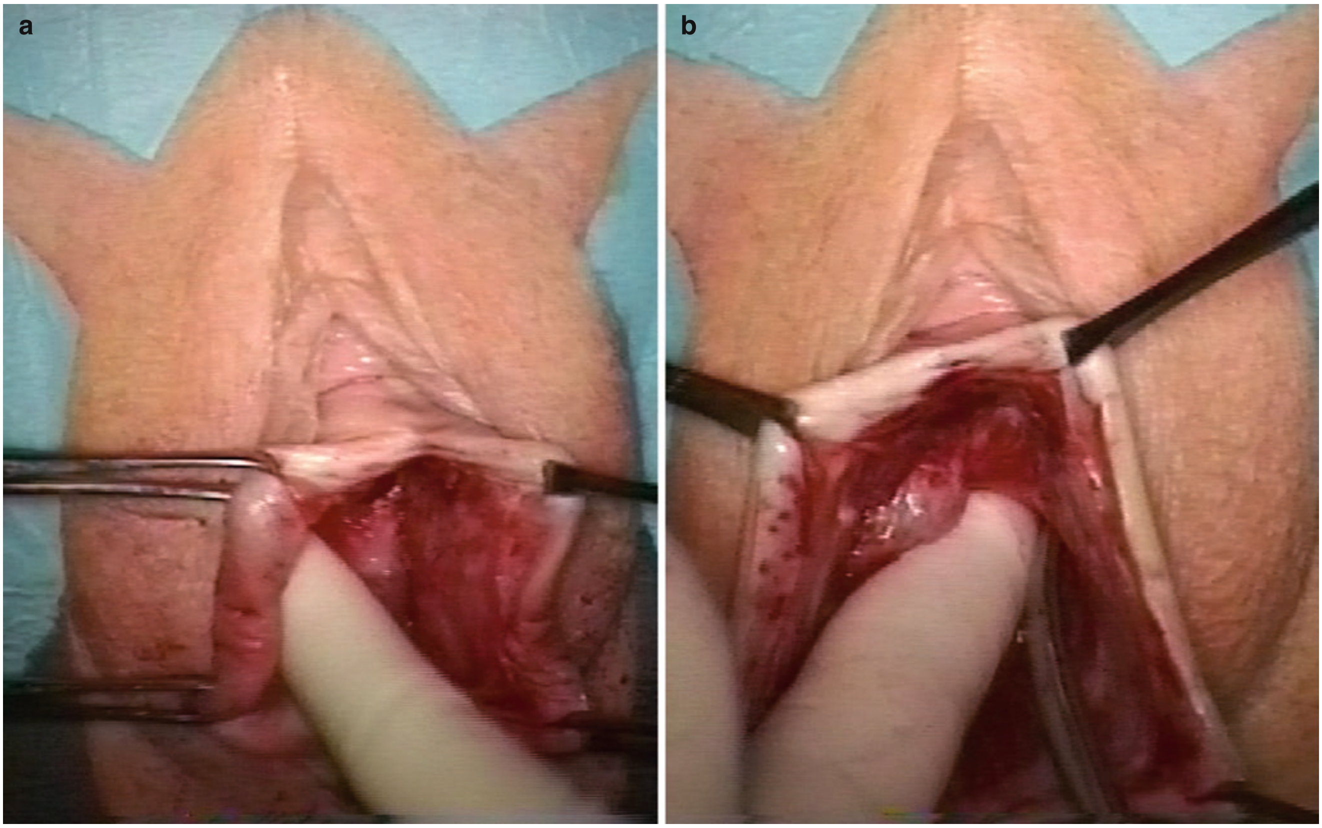

Fig. 13.4 **(a, b)** Opening the paravesical spaces

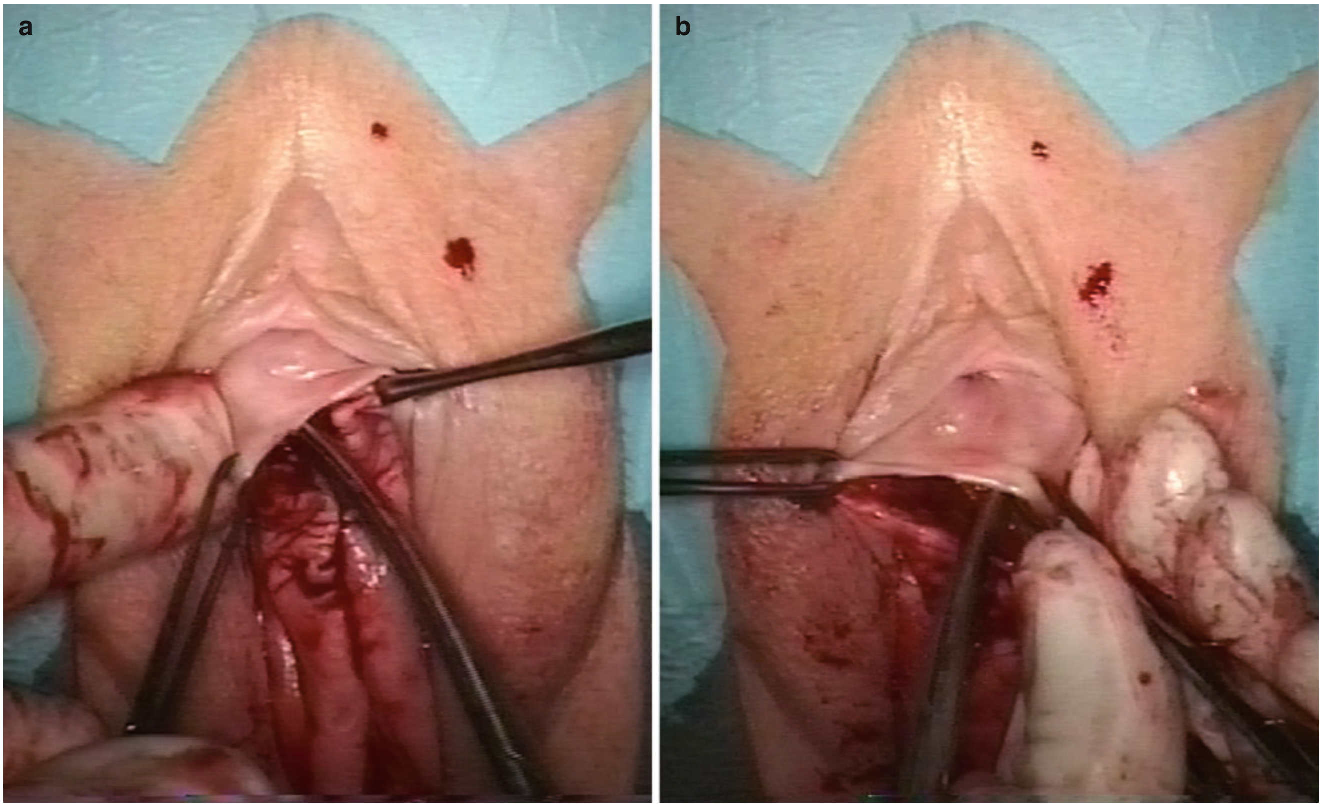

Fig. 13.5 (**a**, **b**) Opening the paraurethral spaces

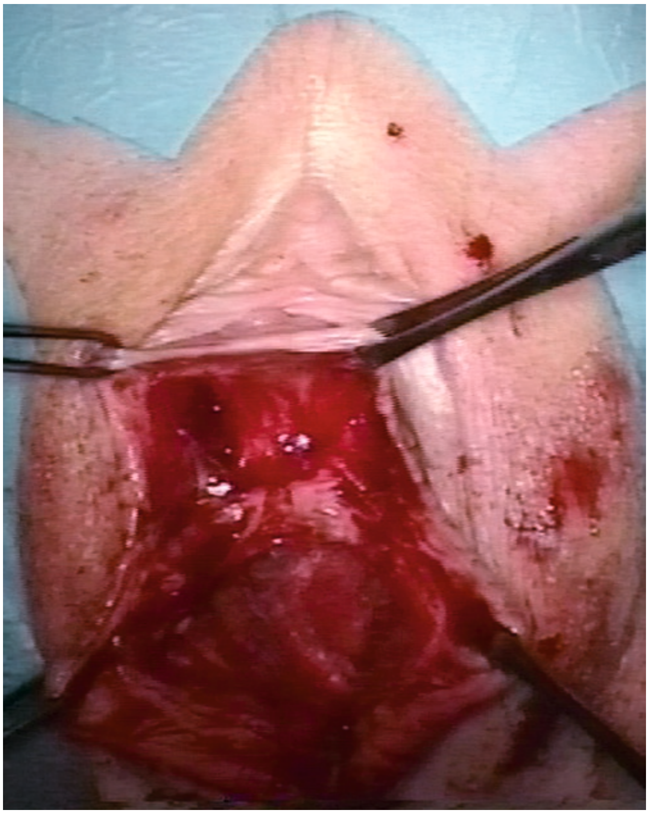

Fig. 13.6 The paraurethral (level 3) and paravesical (level 2) spaces are separated by the pubococcygeus muscle

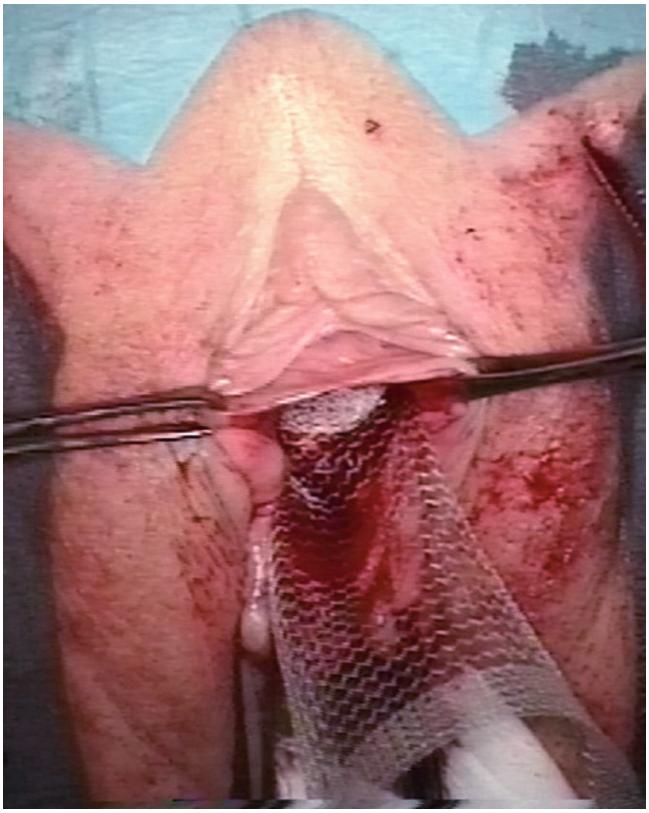

Fig. 13.7 The anterior arm of the Quadra mesh is inserted through the paraurethral spaces (level 3) as any suburethral sling

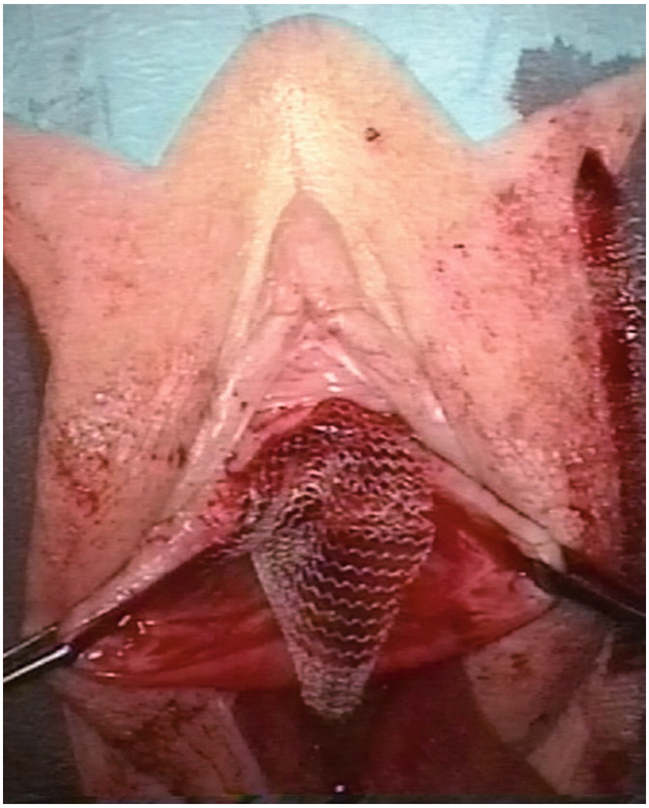

Fig. 13.8 The two posterior arms of the Quadra mesh is inserted through the obturator muscle at the level of the ischial spine in the paravesical fossas (level 2). The mesh is then sutured to the cervix or the vaginal vault

1 cm in the midline. It has to be dissected slightly on 1 cm on each side from the vagina, in order to make the vaginal closure possible above the mesh. It is very important to respect the pubococcygeus muscle as much as possible because it will support the suburethral part of the mesh, preventing any shifting toward the bladder neck. The two anterior arms of the Quadra mesh are inserted with the IVS 04 tunneller through the paraurethral dissection space as for any transobturator suburethral sling, with special care for the absence of tension, treating the SUI. The same tunneller is used for the insertion of the posterior arms. The external obturator muscles are perforated as close as possible to the ischion at the lowest part of the obturator membrane. Then, the blunt tip of the IVS 04 tunneller goes parallel to the internal obturator muscle in direction of the ischial spine and the insertion of the arcus tendineous levator ani. There, the internal obturator muscle and its fascia are perforated and the tip of the tunneller led out of the vaginal incision. The posterior arm is threaded in and passed through the tunnel on both sides. The posterior part of the mesh is sutured to the uterine cervix or to the vaginal vault (if there is no more cervix) with one or two absorbable sutures. The posterior arms are put under tension and the mesh stretches posterior as a subvesical hammock, treating the cystocele. The mesh is wrapped around the pubococcygeus muscle, preventing a direct contact with the vesical trigone. Finally, the vaginal epithelium is sutured without any colpectomy and the running suture closing the fascia at the same time. A vaginal pack and a Foley catheter are inserted for 24 h. Discharge is allowed after 24–48 h.

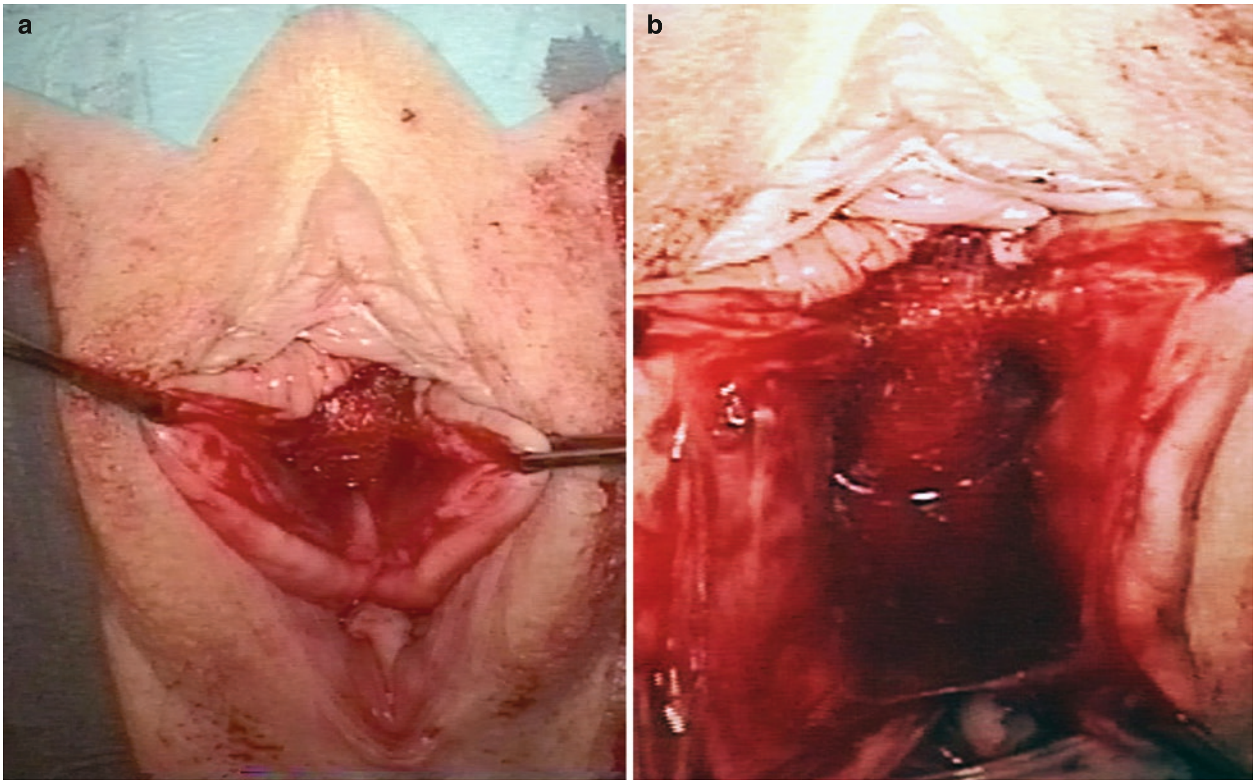

Fig. 13.9 **(a, b)** The suburethral sling and the subvesical hammock are in place

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Part IV

Mid-Compartment Repair

14

Surgical Mesh Reconstruction for Post-hysterectomy Vaginal Vault Prolapse

Giacomo Novara, Walter Artibani, Silvia Secco, and Menahem Neuman

Introduction

According to the 2002 standardization of terminology of the International Continence Society, post-hysterectomy vaginal vault prolapse (PHVVP) is defined as any descent of the vaginal cuff scar after hysterectomy below a point which is at least 2 cm. less than the total vaginal length above the plane of the hymen.[1]

The vaginal vault prolapse might be isolated or combined with prolapse of the anterior or posterior vaginal wall at various degrees. The true prevalence of PHVVP is unclear. The reported prevalence rates ranged from 0.2% to 43%[2,] depending on definitions and accuracy of the patients evaluations. However, figures as high as 10% are usually considered to be more realistic.[3]

The levator ani muscles are the most important muscles of support in the pelvis and their contractions keep the urogenital hiatus closed, preventing any opening in the pelvic floor through which prolapse may occur. When the muscles relax during micturition or defecation, the connective tissue attachments of the pelvis support the pelvic organs.[4] According to the classic description of DeLancey, different levels of support act sustaining the vaginal walls. Level I support occurs at the vaginal cuff and consists of the uterosacral and cardinal ligament complexes, which attach uterus, cervix, and upper vagina to the pelvic walls. Level II support occurs at the mid-portion of the vagina, where the supporting connective tissue layers support and separates the bladder anteriorly and rectum posteriorly from the vagina. At level III the vagina fuses directly with the urethra anteriorly, perineal body posteriorly, and levator ani muscles laterally.[5] Although the clear pathophysiologic mechanisms of prolapse are not completely understood, the occurrence of neuromuscular injuries related to obstetrical pelvic floor trauma, obesity, aging, chronic lung disease, or constipation can lead to a failure of muscle support to the urogenital organs. The pelvic ligaments, consequently, have to fully sustain the pelvic organs, which can lead to the occurrence of prolapse, especially in those cases where biomolecular alterations of collagen synthesis, architecture. or biodegradability are present. With regard to PHVVP, surgical factors such as failure to suspend the vaginal apex to the sacrouterine ligaments or suture break down could lead to vault prolapse. Moreover, the preexisting weakness of the pelvic floor, which is often the primary cause of the uterus prolapse that lead to the indication for hysterectomy, might be responsible for PHVVP.[6–9]

Patients with vaginal PHVVP can vary from being asymptomatic to presenting with various complaints including both storage and voiding lower urinary tract symptoms (LUTS), bowel storage and emptying difficulties, dyspareunia, coital difficulties, and other sexual dysfunctions. Specifically, occult stress urinary incontinence and voiding LUTS, including the need to manually reduce prolapse to void can be frequent in patients with bulky, high-grade prolapse[10–13] and, similarly, manual assistance with prolapse reduction may be required for facilitation of defecation. Moreover, especially in case of high grade prolapse, the lump emerging out of the introitus may interfere with even simple daily activities as walking and sitting, negatively affecting the body image and self-esteem of the affected patient.

Accurate diagnosis of the prolapse is crucial for proper design of a comprehensive therapeutic plan. Therefore, obtaining patient's history is the key for understanding the patient's needs and expectations. Pre-interview filling of questionnaire evaluating symptoms and their impact on quality of life such as the short forms of Pelvic Floor Distress Inventory and Pelvic Floor Impact Questionnaire might be highly useful. Then, a pelvic examination under Valsalva maneuver is mandatory, as post-hysterectomy vaginal vault prolapse coexists frequently together with anterior and posterior vaginal wall prolapse. Accurate mapping and grading of prolapse according to the ICS POP-Q system is needed, as well as evaluation of the vaginal mucosa status, presence of evident or occult urine and fecal incontinence.

Moreover, abdominal ultrasound scan might be of benefit to rule out coexisting pelvic organ diseases, while perineal

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ultrasound scan might be useful for a more appropriate staging of the prolapse.

The accurate place of urodynamic studies in terms of pointing the best therapeutic approach and prediction of cure or complication rates is debatable. Although the urodynamic data might enrich the understanding of the individual pathological backgrounds improving the treatment, some authors argue that the benefit is of no clinical value.[14,15] In the presence of fecal storage or voiding abnormality anorectal work-up is indicated.

Surgical Treatment of Post-hysterectomy Vaginal Vault Prolapse

Surgical treatment of PHVVP can be performed according to several different methods and more than 40 reconstructive surgical techniques have been described.[16] Surgical repair can be performed vaginally or abdominally, in that case with retropubic, conventional laparoscopic, or robot-assisted approach. Sacrospinous vault suspension, ileococcygeus muscle fixation, uterosacral ligament fixation, McCall culdoplasty, and posterior intravaginal slingoplasty are the most commonly used vaginal techniques[17], while abdominal sacrocolpopexy is the most common abdominal procedure on the market.

Theoretically speaking, compared to the abdominal approach, vaginal surgery might allow several advantages, including lower morbidity, possibilities of performing surgery under local or regional anesthesia and repairing simultaneously other pelvic defects, shorter operative time, and quicker patients' recovery. On the other hand, presence of orthopedic deformities, concomitant intra-abdominal pathology, and reduced vaginal length might be conditions which could favor an abdominal approach.[4]

For the purpose of the present review, we focused our attention mainly on mesh augmented surgical repairs.

Although the use of synthetic mesh is very popular during inguinal hernia repair with the purpose of increasing the success rate of traditional facial repair, the application of the same concepts in urogynecology surgery has been less properly evaluated and the lack of consistent clinical data from long-term randomized controlled trials make the use of synthetic mesh quite an empirical issue.

The ideal mesh would be chemically and physically inert, noncarcinogenic, mechanically strong, sterile, not physically modified by body tissue, readily available in a convenient and affordable format for use, inexpensive, and have minimal risk of infection and rejection.[18] Although, to date, there are no biologic or synthetic implants that meet all such criteria, macroporous (pore size greater than 75 μm), monofilament, flexible meshes are usually regarded as the best available option. Pore size >75 mm is necessary in order to allow entry of fibroblasts, macrophages, blood vessels, and collagen fibers, allowing prevention of mesh infection and fibrous ingrowth of surrounding tissues. On the other hand, monofilament mesh has to be preferred because multifilament synthetic material has interstices, which might allow bacteria growth unreachable by macrophages.

In pelvic floor reconstructive surgery, common indications for mesh include suboptimal autologous tissue, connective tissue disorder, the need to bridge a gap, concern about vaginal length or caliber, and pelvic floor denervation. On the other hand, accepted contraindications to mesh grafting included host conditions that may compromise the vascular supply to the pelvic floor such as a history of pelvic radiation, severe diabetes, severe vaginal atrophy, and factors that may predispose the patient to infections such as systemic steroid use, or active vaginal infection.[19]

In the last years, moreover, several mesh kits have been introduced in the market with prolapse repair. Specifically, these kits involve the blind passage of insertion needles through small perineal incisions into the obturator foramen and ischiorectal fossa to facilitate the tension-free vaginal placement of mesh or graft. Anterior, posterior, and total Prolift (Ethicon Women's Health and Urology, Somerville, NJ), Perigee and Apogee (American Medical Systems, Minnetonka, MN), Avaulta (CR Bard, Murray Hill, NJ), and IVS Tunneller (US Surgical, Tyco Healthcare Group LP, Norwalk, CT) are the most commonly used kits, which can be applied to repair anterior, posteriorvaginal wall or apex vagina.[18]

Abdominal Sacrocolpopexy

After Lane described the use of synthetic mesh to suspend abdominally a prolapsed vaginal apex to the sacrum[20], the technique of sacrocolpopexy has evolved over the last 4 decades and, currently, it is considered by most surgeons as the gold-standard procedure for vaginal vault prolapse. The most commonly performed method is attaching the posterior vagina to the level of the rectal reflection and the anterior vagina for a distance of 4–5 to 1–2 cm below the sacral promontory. Suture materials and type of mesh remains controversial, but most surgeons seems to agree that permanent materials are essential and polypropylene mesh grafts are usually regarded as the gold standard. Specifically, Culligan et al. reported on 100 patients with HVVP who were randomized to sacral colpopexy with polypropylene mesh or cadaveric fascia lata.[21] At 12-month follow-up, 91% of the patients treated using the polypropylene mesh and 68% of those where fascia lata was used were objectively cured ($p = 0.007$), with significant differences identified in points Aa, C, and POP-Q stage.[21]

Intra- and perioperative complications of abdominal sacrocolpopexy include urinary tract infection (10.9%), wound infections (4.6%), cystotomy (3.1%), enterotomy or proctotomy (1.6%), postoperative ileus (3.6%), thromboembolic events (3.3%) and transfusions (4.4%).[22] Massive bleeding during the presacral dissection, especially if done at the S3–S4 level, might be a major complication, occurring in 1.2–2.6% of the cases. Mesh erosion is reported to occur in up to 5% of cases but larger series and review papers suggested that the rate of erosions with polypropylene was as low as 0.5%.[22,23] Moreover, a secondary analysis of the Colpopexy and urinary reduction efforts (CARE) trial demonstrated that the use of expanded polytetrafluoroethylene mesh (odds ratio 4.2) as well as current smoking (odds ratio 5.2) and concomitant hysterectomy (odds ratio 4.9) may significantly increase the risk for mesh erosion following sacrocolpopexy[24]

Since abdominal sacrocolpopexy is a time-honored procedure, a few retrospective reports at long-term follow-up are available, demonstrating that the success rate 5–10 years after surgery were in the range of 85–97%.[25]

Three randomized controlled trials compared abdominal sacrocolpopexy and vaginal sacrospinous ligament fixation in the treatment of vaginal vault prolapse after hysterectomy.[26–28] In the most methodological accurate one, Maher et al. randomized 95 patients to abdominal sacrocolpopexy or vaginal sacrospinous colpopexy. Specifically, the study included the use of several validated questionnaires, such as the Short Urinary Distress Inventory, the Incontinence Impact Questionnaire, and the Short-Form 36 Health Survey. After a mean follow-up of 2 years, both subjective (94% in the abdominal arm vs 91% in the vaginal arm) and objective cure rates (76% vs 69%, respectively) were quite high and similar for both procedures.[26] As far as emptying and voiding lower urinary tract symptoms were concerned, a prior frequency–urgency syndrome was cured in 27% of the patients who underwent abdominal surgery and in 37% of those in whom vaginal surgery was carried out, while de novo frequency–urgency syndrome occurred in 34% and 22% of the patients, respectively. Preoperative voiding dysfunction was cured in about 80% of the patients in both arms, while de novo voiding symptoms were shown only in a few patients. Bowel function, evaluated in terms of postoperative constipation (36% vs 27%), obstructed defecation (9% vs 6%), and fecal incontinence (4% vs 8%), were similar in both arms. Preoperative dyspareunia resolved in 56% of the patients randomized to the abdominal surgery arm and in 43% randomized to vaginal surgery, being present postoperatively in about 20% of the cases. In both arms, the scores of Short Urinary Distress Inventory and Incontinence Impact Questionnaire were similar and significantly improved after surgery. Vaginal sacrospinous colpopexy, however, was followed by significantly higher risks of anterior vaginal wall and vault prolapse (45% vs 13% in the abdominal surgery group) but abdominal sacrocolpopexy was associated with a longer mean operating time (106 ± 37 vs 76 ± 42 min; $p < 0.01$), slower return to activity of daily living (31 ± 12 vs 25.7 ± 9.7 days; $p < 0.01$), and greater costs[26]. The results of all the 3 RCTs were combined in a Cochrane meta-analysis, where abdominal sacrocolpopexy was found to outperform vaginal sacrospinous colpopexy for recurrence of vaginal vault prolapse (3% vs 16%; relative risk [RR] 0.23, 95% CI 0.07–0.77; $p = 0.02$); postoperative SUI (19% vs 34%; RR 0.55, 95% CI 0.32–0.95; $p = 0.03$), dyspareunia (15% vs 36%; RR 0.39, 95% CI 0.18–0.86; $p = 0.02$). However, abdominal sacral colpopexy was associated with longer operating time (weighted mean difference [WMD] 21 min, 95% CI 12–30; $p < 0.00001$), longer time to recover (WMD 8.3 days, 95% CI 3.9–12.7; $p < 0.05$) and was more expensive (WMD US$1,334, 95% CI 1,027–1,641; $p < 0.00001$) than the vaginal approach.[29] Finally, the two techniques yielded similar rates of surgery-related adverse events, hospital stay duration, and the need of repeated surgery for prolapse or SUI.[29]

On the whole, since that vaginal sacrospinous colpopexy is quicker and cheaper to perform and women have an earlier return to activities of daily living, abdominal sacrocolpopexy might be more indicated for young and active women who accept longer recovery and the potential risk of foreign body erosion to achieve higher success rate.

Laparoscopic Sacrocolpopexy

Laparoscopic pelvic floor reconstructive surgery aims at maximizing the efficacy of abdominal pelvic floor reconstructive surgery, reducing the perioperative morbidity and shortening the in-hospital stay. Theoretically, all pelvic surgery performed through a laparotomic access can also be executed laparoscopically.

Although no randomized comparison of open versus laparoscopic sacrocolpopexy has been published, a few authors reported retrospective comparative studies.

Specifically, Hsiao et al. recently evaluated 25 patients undergoing laparoscopic and 22 abdominal sacrocolpopexy.[30] The authors found that mean estimated blood loss ($p = 0.0002$) and mean length of hospitalization ($p < 0.0001$) were significantly lower after laparoscopic surgery, although operative time was significantly longer (219.9 vs 185.2 min, $p = 0.045$). Finally, success rates were similar for both procedures (100% after laparoscopic surgery at 5.9-month follow-up versus 95% after abdominal surgery at 11-month follow-up).[30] Quite similar data, moreover, were reported by Paraiso et al.[31] and by Klauschie et al.[32]

With regard to the outcome of laparoscopic sacrocolpopexy, in the largest published prospective studies, Sarlos et al. recently reported on a series of 101 patients evaluated at 12-month follow-up. The median duration of surgery was 141 min and mean blood loss was 95 mL, with no patients receiving blood transfusion. Only two procedures were converted to laparotomy, and intraoperative complications included three cases of rectal injuries (one repaired laparoscopically, one laparotomically, and one diagnosed during the postoperative day 2 due to septical peritonitis, which require laparotomy with sigmoidostomy); four cases of bladder lesions repaired laparoscopically, which needed a prolonged catheterization; a single case of bleeding from epigastric vessels occurred during trocar placement managed laparoscopically. A single major postoperative complication occurred, that is, a mechanical ileus due to adhesions, which required laparotomy during postoperative day 4 where adhesiolysis and resection of a bowel segment were performed. At 12-month follow-up, the subjective and objective cure rates were 98% and 92%, respectively. Specifically, no patients had apex vaginal prolapse, while six patients had anterior and two had posterior vaginal wall prolapse, with only two of them being symptomatic and a single one undergoing anterior colporrhaphy with vaginal mesh augmentation. With regard to functional results, 24 (24%) patients developed de novo stress incontinence and 15 (15%) of them underwent further anti-incontinence surgery, while the most frequent complaint was constipation, present in 18 (18%) patients during the first 6 months after surgery.[33]

Further data at longer follow-up are available from two retrospective studies. Specifically Ross et al. reported on 43 patients, demonstrating a 93% objective cure rate at 5-year follow-up.[34] Similarly, Higgs et al. demonstrated in a series of 64 patients evaluated at a median follow-up of 66 months that 42% of the patients had POP-Q stage 0, 20% stage I, 32% stage II, and 6% stage III prolapse, with only four cases of recurrent vaginal vault prolapse, with 16% of the patients undergoing further surgery for recurrent prolapse. Interestingly, the authors reported an erosion rate of 6% in case of non-vaginally placed mesh.[35]

On the whole, although functional long-term data on recurrent prolapse and functional outcomes were not sufficiently reported, laparoscopic sacrocolpopexy seems to yield anatomic results similar to the abdominal approach. Although laparoscopic approach requires longer operating time, it can allow significantly shorter hospital stay. However, technical difficulties of a surgical procedure requiring extensive laparoscopic dissection and considerable skills in suturing limit the widespread diffusion of laparoscopic sacrocolpopexy.

Robotic-Assisted Laparoscopic Sacrocolpopexy

The shift from open to laparoscopic surgery represents a completely new experience for surgeons, who have to learn a new surgical anatomy and new operative procedures and must deal with new surgical tools. More specifically, the reduction of the range of motion (with only 4 degrees of freedom), two-dimensional vision, the impaired eye–hand coordination (misorientation between real and visible movements), and the reduced haptic sense with only minimal tactile feedback provided by laparoscopic tools are the main restrictions associated with a steep learning curve.[36] Robotic systems have recently been introduced in an attempt to reduce the difficulty of performing complex laparoscopic procedures, particularly for nonlaparoscopic surgeons, and it is gaining widespread diffusion in urology, especially in urologic oncology, with laparoscopic radical prostatectomy being the most common robotic-assisted procedure worldwide. Specifically, the da Vinci robot includes a true three-dimensional imaging system, which provides magnification up to 12-fold, and the Endowrist technology, which provides 7 degrees of freedom, duplicating the dexterity of the surgeon's forearm and wrist at the operative site. There are major advantages for suturing, which makes the robotic technology suitable for pelvic floor reconstructive surgery.[36,37]

Clearly, the purpose of robotic laparoscopy is to provide durable repair of POP, reducing postoperative pain and complications through the use of a laparoscopic technique, shortening the learning process by the use of the robotic system. Technically speaking, robotic sacrocolpopexy aims at reproducing the steps of abdominal and pure laparoscopic sacrocolpopexy. The patient is placed in the dorsal lithotomy position and through a transperitoneal access one camera port, two robotic ports, and two standard laparoscopic ports are placed. The daVinci robot is used to mobilize vagina and visualize sacral promontory, perform a standard laparoscopic dissection in combination with an intravaginal retractor, and suture a mesh graft from the vagina to the sacral promontory, as well as for culdoplasty, and retroperitonealization of the graft.[38]

To date, two series have been published describing experience with robotic-assisted laparoscopic sacrocolpopexy (RASC) in treatment of post-hysterectomy vaginal vault prolapse. Elliott et al. from Mayo Clinic[39] reported on 30 patients with post-hysterectomy vaginal vault prolapse treated with RASC. Mean operative time was 3.1 h (range 2.15–4.75). Only a single procedure was converted to open abdominal surgery due to unfavorable anatomy and all patients but one were discharged from the hospital after an overnight stay. Postoperative complications included only two cases of port site infections. At a mean follow-up of 24 months, all patients reported to be satisfied with the outcome of their surgery and all of them but one would recommend the same procedure to a friend. A single patient developed recurrent vaginal vault prolapse, which was treated by abdominal sacrocolpopexy, while another patient had a high-grade posterior vaginal wall prolapse, which was treated by posterior colporrhaphy. Six months after surgery, two more patients developed small vaginal erosions of the mesh at the level of the vaginal cuff, which were treated with transvaginal excision of the mesh and primary closure, without any further sequelae.[39]

Daneshgari et al. recently reported the Cleveland Clinic experience with RASC.[40] Specifically, 15 women with stages III or IV POP involving the apical, anterior, and\or posterior wall were treated, with seven patients having concurrent placement of a transobturator tape sling and one patient Burch colposuspension. The mean operative duration was 317 (258–363) min and the mean estimated blood loss during surgery was 81 (50–150) mL. Conversion to laparotomy was required in 3 cases, while one patient had an intraoperative serosal bladder injury during division of dense adhesions, which was recognized and repaired immediately without subsequent morbidity. There were no postoperative wound infections or separation. The mean hospital stay was 2.4 (1–7) days. At a mean follow-up of 3.1 months, all patients had POP-Q stage 0 prolapse, with significant improvements in POP-Q values. Specifically, the postoperative POP-Q values were the following: Aa and Ba – 2.29 cm; Ap and Bp – 2.65 cm; C – 8.28.[40] On the whole, the two studies had the major merit of standardizing the technique of RASC, demonstrating that the technique was feasible with good short-term anatomical outcome. However, the limited number of enrolled patients, short follow-up duration, and lack of functional results related to bowel, bladder, and sexual function prevent us from drawing definitive conclusions on the technique.

Geller et al. very recently reported a nice retrospective comparative study, evaluating robotic and abdominal sacrocolpopexy.[41] Specifically, the authors compared 73 patients treated robotically and 105 having traditional open surgery. With regard to the perioperative data, robotic surgery was associated with significantly lower intraoperative blood loss (103 ± 96 vs 255 ± 155 mL; $p < 0.001$), and shorter length of stay (1.3 ± 0.8 days vs 2.7 ±1.4 days; $p < 0.001$), although operative time was significantly longer (328 ± 55 vs 225 ± 61; $p < 0.001$). Moreover, at 6-week follow-up evaluation, slight improvement in C point was found following RASC (–9 vs –8 following open sacrocolpopexy; $p < 0.008$), with no difference in other POP-Q points.[41] However, the methodological design of the study (retrospective comparison with historical series of open sacrocolpopexy), short-term follow-up, and lack of validated questionnaires to assess functional outcome prevent us from drawing definitive conclusions on the comparisons of open and robotic sacrocolpopexy.

Clearly, robotic surgery had the major limitations of costs, especially for robot purchase, maintenance and operative equipment per case, which usually overshadowed shorter hospital stay and makes robotic sacrocolpopexy more expensive than both abdominal and conventional laparoscopic surgery.[42] However, costs of robotics are volume-dependent and, consequently, this technology is possible in high-volume centers. However, urologists are becoming more and more familiar with the robotic approach to radical prostatectomy and can use their experience to expand the indications and benefits of robotic surgery to sacrocolpopexy. Another potential limitation to the diffusion of RASC might be the lack of adequate training opportunities, lack of expertise surgeons in communities to help further advance the skills of younger surgeons, and the thought that long learning curves to develop skills are required.[43] The development of computer-based simulators will allow surgeons in the future to learn the skills required to manipulate the robot before it operates with a live patient. However, the burden of experiments done by urologists in the treatment of prostate cancer with robotic radical prostatectomy can provide a significant and precious background for RASC, which is a procedure requiring extensive suture skills.

Mesh Kit

The available literature on mesh kits was quite limited, due to the recent diffusion of such procedures. In one of the largest series, Neuman evaluated 140 patients with apex vagina prolapse undergoing posterior IVS. Intra- and perioperative complications were quite acceptable, with a 4% rate of pelvic hematoma. At follow-up, tape erosions were identified in 13 patients (9.3%), with tape resection needed in most of the cases. Anatomical results were quite good, with only three patients (2%) presenting with recurrent vaginal vault prolapse and four (2.9%) with anterior or posterior vaginal wall prolapse.[44]

Higher quality evidence was recently provided by some other studies. De Tayrac et al. recently published an interesting randomized controlled trial, comparing mesh kit and sacrospinous suspension.[45] Specifically, 49 patients with uterine or PHVVP were randomized to infracoccigeal sacropexy (IVS Tunneler) or traditional vaginal repair by sacrospinous suspension. IVS turned out to be quicker (13.2 ± 5.2 vs 20 ± 8.1 min, $p = 0.002$), easier than sacrospinous fixation, and no significant intraoperative complications were observed in both arms. During postoperative day 1, mean level of pelvic or buttock pain was significantly lower in those patients having IVS (VAS scale 1.3 ± 1.6 vs 3.2 ± 2.7, $p = 0.005$). At a mean follow-up of 16.8 months, the anatomical results of both procedures were pretty similar, as well as reoperation rates (two cases in each arm due to erosions or anterior vaginal wall mesh, and a single case in each arm due to uterine prolapse and anterior vaginal wall prolapse, respectively). Moreover, the study uses Pelvic Floor Distress Inventory, Pelvic Floor Impact Questionnaire, and Pelvic Organ Prolapse–Urinary Incontinence–Sexual Function Questionnaire to evaluate the prolapse-related symptoms and functional outcomes after prolapse repair, which finally were overlapping in both arms.[45]

Quite similar data, moreover, were provided in another randomized controlled trials (to date, presented as a congress abstract but not published on peer-reviewed journals) by Meschia et al.[46]. Specifically, the study randomized 66 patients with PHVVP to posterior IVS or sacrospinous fixation, demonstrating at a median follow-up duration of 19 and 17 months overlapping figures in terms of anterior (27% in the IVS vs 33% in the sacrospinous fixation arm, $p = 0.89$), posterior (18% vs 12%, $p = 0.80$) vaginal wall prolapse recurrences, with a single patient in the IVS group experiencing recurrent PHVVP. Two cases of mesh erosion and a single case of perirectal abscess were observed in the IVS arm.[46]

On the whole, the two studies suggested that IVS was as effective as sacrospinous fixation at short-term follow-up, although IVS procedure was quicker and probably followed by slightly lower postoperative pain. However, randomized trials with long-term outcome are highly desirable.

Further interesting data, moreover, were provided by two recent systematic reviews of the literature.[47,48] Specifically, Feiner et al. reported a systematic review of efficacy and safety of mesh kits in the treatment of apex vaginal wall prolapse.[47] The authors identified 30 studies, including 20 congress abstracts and 10 papers published on peer-reviewed journals, evaluating more than 2,600 patients. Finally, the authors estimated success rates as high as a 95.4% for Apogee (at a mean follow-up of 26 ± 15 weeks), 86.8% for Prolift (at a mean follow-up of 30 ± 12 weeks), and 88.2% for posterior IVS (at a mean follow-up of 46 ± 36 weeks).[47] In the other systematic review focused on complications, the same group of authors evaluated 24 studies collecting more than 3,400 patients, reporting an overall 14.5% complication rate for mesh kit at a mean follow-up of 17.1 ± 13.8 months, including a 5.8% of mesh erosion. Specifically, 8.5% were grade 3 complications according to the Dindo classifications, which required surgical intervention to be treated.[48] In comparison with the figures of traditional vaginal and abdominal repair reported in the meta-analysis, mesh kits seem to expose the patients to a similar risk of overall complications (15.3% and 17.1% in meta-analyzed papers evaluating vaginal and abdominal procedures, respectively), although the estimated rates of grade 3 complications were significantly lower in traditional vaginal (1.9%) and abdominal (4.8%) repairs. On the contrary, reoperation rates for recurrent POP were significantly lower in the patients treated with mesh kit (1.3%, vs 3.9% of traditional vaginal repair, and 2.3% of abdominal sacrocolpopexy) although the mean follow-up of the published reports evaluating mesh kits were significantly lower.[48] Although those data do not have the value of a randomized controlled trial and most of the included publications on mesh kits were conference abstracts, on the whole the figures of the two systematic reviews suggested that success rates of mesh kits was quite high, as evaluable at the follow-up durations of the available studies. Although total complication rates seemed similar for traditional vaginal surgeries, sacral colpopexy, and vaginal mesh kits for the treatment of apical prolapse, however, the reoperation rate due to complications was highest in the vaginal mesh kit group, despite the shortest follow-up period. Although those data might at least partially reflect the learning curve of new surgical procedures, indeed, they seem to support the recent FDA public health notification about serious complications associated with transvaginal placement of surgical mesh in repair of pelvic organ prolapse, suggesting the need for specialized training for each mesh placement technique, and awareness of its risks.[49]

Conclusion

According to the most consistent available pieces of evidence, abdominal sacrocolpopexy offers lower risk of recurrent vaginal vault prolapse, postoperative stress urinary incontinence, and dyspareunia; but those results are achievable at the cost of longer operating time, longer time to recover, and higher cost, compared to sacrospinous fixation. Laparoscopic sacrocolpopexy seems to yield anatomic results similar to the abdominal approach but with significantly lower perioperative morbidity and shorter hospital stay, although long-term data on anatomic and functional outcomes are needed to draw clear conclusions. Due to the technical difficulties and steep learning curve of conventional laparoscopic sacrocolpopexy, robotic-assisted laparoscopic sacrocolpopexy might be of interest in order to shorten the learning curve, especially in those centers using robotic surgery for other indications. However, although promising, the available data are very preliminary and concerns about costs and training opportunity are very reasonable, especially among those specialists who cannot benefit from the experience in robotic surgery for prostate cancer treatment. Mesh kits were shown to have anatomic success rate similar to sacrospinous fixation, making the procedure a very interesting option for a minimally invasive treatment of patients with

PHVVP. However, the risk of erosions of the vaginally placed mesh, and reoperation rate due to such complications are consistent. In order to minimize the risk of complications due to the blind steps of the procedures, such kind of surgery requires a clear and comprehensive knowledge of the anatomy and specialized training and further high-quality evidence are, however, needed.

Although the choice of the approach should be based on what is the best for patient's individual variables, the experience of the surgeon and his/her opinion can obviously steer patients to a kind of surgical approach and factors such as previous reconstructive procedures, importance for sexual function, vaginal length, medical comorbidities, tissues quality, associated colorectal problems must be taken into account.

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15

Is Hysterectomy Necessary to Treat Genital Prolapse?

Mohamed Hefni and Tarek El-Toukhy

Is hysterectomy necessary to treat genital prolapse? The aim of any surgical repair procedure is to preserve and maintain the function of healthy organs while minimizing morbidity. To understand the place of vaginal hysterectomy in the treatment of genital prolapse, it is essential to review the history of its development and the management of genital prolapse. It is also vital to review a summary of the recent knowledge of anatomy in relation to pelvic floor support.

History

Historically, the first true vaginal hysterectomy (VH) was described in 1521 by Berengarius de-Capri as a treatment for uterine prolapse.[1] At this time, uterine support and pelvic dynamic anatomy were not yet known, so the idea that if the uterus was coming down it should be removed was acceptable then. In the sixteenth century, several devices were developed for the treatment of genital prolapse, such as oval-shaped pessaries made of hammered brass and waxed cork, and an apparatus made of gold, silver, or brass, which were kept in place by a belt worn around the waist.

Interestingly, management of genital prolapse has not changed much since the sixteenth century. Over the following few centuries, de-Capri's technique has evolved into our present technique in which the cardinal-uterosacral ligaments are shortened and sutured into the vaginal vault after removal of the uterus. In addition, a large number of pelvic organ prolapse repair procedures have been described with varying success rates reported for each procedure.[2,3]

It is estimated that women have an 11% life-time risk of undergoing surgery for pelvic organ prolapse.[4] This rate is projected to increase over the next 2–3 decades.[5,6]

The search for the optimum surgical procedure to correct uterovaginal prolapse has faced many challenges, including lack of clear understanding of the role played by different pelvic structures in the initiation and propagation of pelvic organ descent, poor standardization of reporting symptoms and examination findings before and after surgery and the small sample size, and high drop-out rate and lack of objective long-term results in most published studies.[2,3]

Anatomical Considerations

There is no doubt that recent anatomical studies of pelvic floor support and understanding of pelvic dynamics will eventually lead us beyond the current management of pelvic floor defects. As we are now able to identify the specific defect (or defects) responsible for genital prolapse, it is possible that specific procedures may be developed and used to address these individual defects.

DeLancey's[7,8] anatomical cadaver studies have shown that pelvic organs are suspended by the pelvic ligament and supported by the levator ani muscle. Breaks in the connective tissue and neuromuscular damage affecting the pelvic floor muscle cause pelvic organ prolapse. Magnetic resonance imaging (MRI) and ultrasonography have begun to define the dynamics of the pelvic floor and document specific tissue lesions involved in this process.

The structures that support the vagina and the uterus are divided into three levels[7], which correspond to differing areas of support (Table 15.1).

Level 1 (Suspension)

The upper part of the vagina and the cervix are suspended from above. The suspending structure that is attached to the uterus is called the parametrium and that attached to the vagina is the known as the paracolpium. The parametrium is made up of what is clinically referred to as the cardinal and uterosacral ligaments, and continues down the vagina as the paracolpium. The upper portion of the paracolpium is responsible for suspending the apex of the vagina after hysterectomy.

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Table 15.1 The three levels of support

Level	Structure	Function	Effect of damage
Level I: suspension	Parametrium and paracolpium	Suspends uterus and upper vagina	Uterine prolapse or vault prolapse
Level II: attachment	Pubocervical fascia Rectovaginal fascia	Supports bladder Supports rectum	Cystocele–urethrocele Rectocele
Level III: fusion	Levator ani and perineal body	Fixes vagina to adjacent structures	Urethrocele or perineal deficiency

Level 2 (Attachment)

In the middle portion of the vagina, the paracolpium becomes shorter and is attached medially to the vaginal wall and laterally to the pelvic side walls.

Level 3 (Fusion)

This corresponds to the region of the vagina that extends 2–3 cm above the hymenal ring; the vagina is fused laterally to the levator muscle and posteriorly to the perineal body, while anteriorly it blends with the urethra.

The opening within the levator muscle through which the urethra and the vagina pass (and through which the prolapse occurs) is called the urogenital hiatus of the levator ani. The hiatus is bound anteriorly by the pubic bone, laterally by levator ani muscle, and posteriorly by the perineal body and external anal sphincter. It has been demonstrated that increasing pelvic organ prolapse is associated with increased urogenital hiatus size.[9] Furthermore, the hiatus was found to be larger after several failed repair operations than after successful surgery or a single failure.

Damage to the upper suspensory fibers of the parametrium and paracolpium causes a different type of prolapse from damage to the mid-level support of the vagina[8]. Therefore, while the loss of the upper suspensory fiber of the paracolpium and the parametrium is responsible for the development of uterine prolapse and vault prolapse, the defects in the support provided by mid-level vaginal support (pubocervical and rectovaginal fasciae) result in a cystocele and/or rectocele. The support under the urethra has special importance for urinary incontinence. These defects usually occur in varying combinations.

As these specific defects will lead to certain types of prolapse, specific surgical procedures will be needed, for example, if there is a defect at level 2 with detachment of the pubocervical fascia, it will result in the presence of a cystocele; it would be a mistake to believe that the attachment of the vaginal vault to the sacrospinous ligament would correct the anatomical defect of the anterior vaginal wall at level 2. On the other hand, if the parametrium or paracolpium is overstretched resulting in second-degree uterine prolapse or vault prolapse, anterior vaginal repair will not correct this type of prolapse and only suspension of the vagina vault will correct such a defect.

Vaginal Axis

A study using MRI[10] demonstrated the function and actual shape of the levator ani. This study showed that the levator ani muscle was dome-shaped at rest. During voluntary pelvic contractions; it straightened becoming more horizontal and, during bearing down, it descended becoming basin shape. This MRI study and others have demonstrated the importance of the vaginal axis over the levator ani plate; in particular during increased intra-abdominal pressure. Not only does the tone of the levator muscle increase during increased intra-abdominal pressure, but the configuration of the muscle is also altered – it is straightened and made more horizontal to support the vagina. Colpography has demonstrated that the upper vagina lies on an almost horizontal axis toward the sacrum.[11] Using vaginography, Funt et al.[12] and Delancey[13] have also confirmed an angulated shape of the normal upper vagina and that the angle between the upper and lower vaginal axis is about 130° (Fig. 15.1). After hysterectomy, the

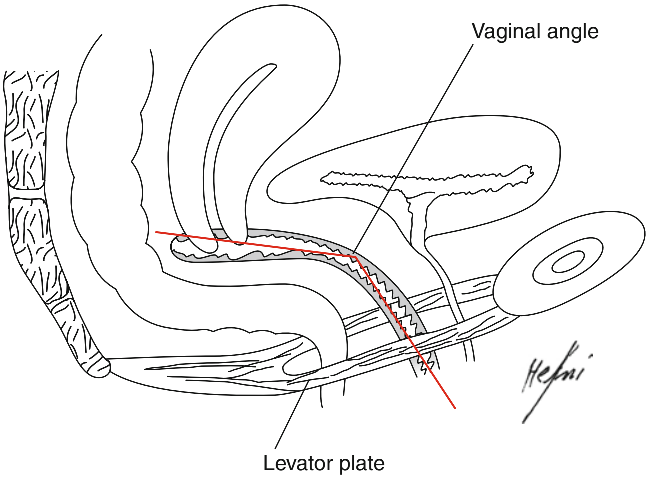

Fig. 15.1 The axis and angle of normal vagina (From Hefni[14])

upper third of the vagina is suspended by the vertical fibers of the paracolpium. However, if these fibers are damaged then vault prolapse might occur.

Sacrospinous Ligament

The sacrospinous ligament is a fibromuscular structure arising from the ischial spine, which fans out and inserts into the lower lateral aspect of the sacrum. The ligament has very distinctive characteristics on palpation; the superior margin of the ligament is hard like bone and the surface of the ligament is corrugated. The inferior margin of the ligament is soft and can be flicked with the finger. There are important anatomical structures in relation to the ligament (Fig. 15.2). The pudendal nerve and vessels run just behind the ischial spine. The inferior gluteal vessels run about a centimeter above the superior margin of the ligament. The sacral plexus and sciatic nerve are located above the superior margin of the ligament. The rectal venous plexus runs at the medial border of the ligament and surrounds the rectum.

Miyazaki[15] clarified the anatomy of the sacrospinous ligament by cadaver dissection to find that the coccygeus muscle and sacrospinous ligament are one structure, and that the sacrospinous ligament was attached directly to the underlying structure of the sacrotuberous ligament. There was no separate distinct sacrospinous ligament lying between the coccygeus muscle and sacrotuberous ligament. Posterior to the sacrospinous ligament lies the gluteus maximus muscle superiorly and the fat of the ischiorectal fossa inferiorly. There were no major blood vessels or nerves running through the ligament, and Miyazaki concluded that the region posterior to the sacrospinous ligament was safe to penetrate and insert a suture.

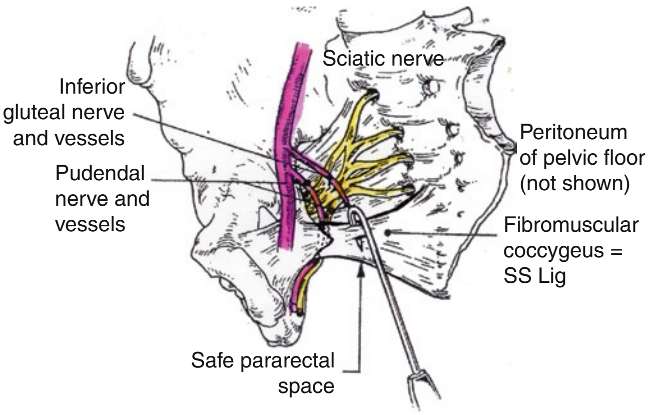
Sciatic nerve
Inferior gluteal nerve and vessels
Pudendal nerve and vessels
Peritoneum of pelvic floor (not shown)
Fibromuscular coccygeus = SS Lig
Safe pararectal space

Fig. 15.2 The anatomy of Sacrospinous ligament

Methods to Correct Uterine Prolapse (Level 1 Defect)

Vaginal Pessaries

Vaginal Pessaries are useful as a temporary measure while waiting for surgical correction or for women who have medical conditions that make them unsuitable for anesthetics. Otherwise, vaginal pessaries are unacceptable as a long-term treatment strategy particularly in sexually active women.

Vaginal Hysterectomy

> *Because the pathologic descent of the uterus is the result of genital prolapse, hysterectomy should not be the prime objective of surgery for genital prolapse. For the patient who wishes to retain her uterus, the surgeon may elect to perform colpopexy without hysterectomy*
>
> *David Nichols*[16]

Vaginal hysterectomy (VH) has traditionally been considered an integral step of the repair procedure[17-19] due to the perceived advantage that hysterectomy facilitates pelvic floor repair and improves results.[20]

In recent years, a shift in our understanding of the dynamics of pelvic organ support[7,8] and the need to reduce surgical morbidity in an aging population have led researchers to question the role of VH in uterovaginal prolapse repair [21-23]. In addition, an increasing number of women are declining hysterectomy because of delaying childbearing to a later age, the perception that the uterus is necessary for sexual satisfaction and the desire to avoid major surgery.[24] In addition, there are two major disadvantages that often occur after VH. One is the high risk of subsequent of vault prolapse and the other is that the vagina is usually left unduly shortened.

Cervical and Uterine Suspension

Several techniques have been reported with acceptable success rates. These include vaginal sacrospinous cervicocolpopexy, vaginal posterior intravaginal slingplasty (IVS), abdominal or laparoscopic sacrocolpopexy, and posterior Mesh repair. The sacrospinous ligament suspension of the uterus is called sacrospinous cervico-colpopexy, or sacrospinous hysteropexy. It is also known as sacrospinous fixation (SSF) of the uterus. We will be using the abbreviation of "SSF" in our text.

SSF is the operation of choice for the management of uterovaginal prolapse at Benenden Hospital and since other techniques have been described elsewhere in this book, we will only describe the technique of SSF. We must, however, stress that the other techniques are equally good techniques for the treatment of uterine prolapse.

Surgical Technique of Vaginal Sacrospinous Cervico-Colpopexy (SSF)

The operation is performed with the patient in the lithotomy position. A size 12 Foley's catheter is fixed and a perineal pouch (Steri-drape, 3 M, and St. Pauls, Minnesota) is used to collect blood. The uterine supports and vaginal wall defects are first assessed. This is performed by grasping the posterior cervical lip with an Allis's forceps (Fig. 15.3) and placing its tip 1–2 cm medial to the right ischial spine. Assessment of the anterior vaginal walls is carried out at this stage to determine if a cystocele is present, which should be repaired first.

The incision is either made from 2 cm below the cervix in the upper half of the vagina or, if the patient requires perineorrhaphy, it starts from the perineum all the way up to the about 2 cm from the cervix. If the latter is required, the hymenal margin is grasped with two Allis's forceps and, by bringing the tips of the forceps together, it is possible to determine the extent of dissection required to prevent dyspareunia in the future. The posterior vaginal wall is infiltrated (Fig. 15.4) with adrenaline and saline (1:200,000) to facilitate dissection between the vagina and the rectum.

A triangular piece of skin is then excised, the base of the triangle being between the tips of the Allis' forceps and the apex toward the anus (Fig. 15.5); then the rectovaginal space is dissected using a knife to expose the transverse perineal muscle at the lower part of the vagina (Fig. 15.6).

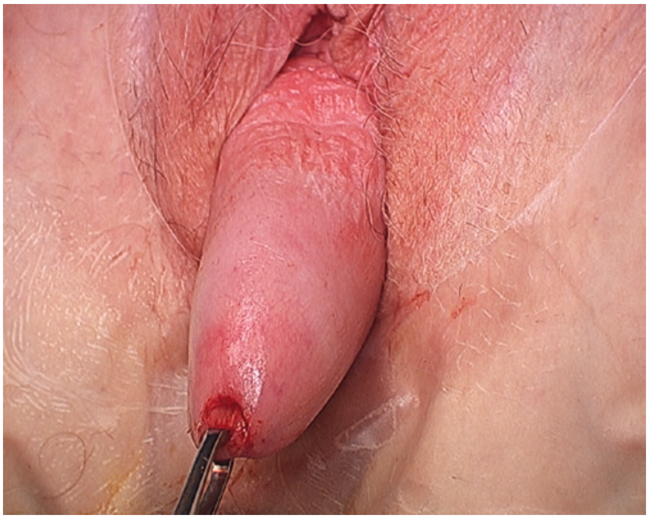
Fig. 15.3 Allis's forceps holding the posterior lip of the cervix. Second degree uterine prolapse

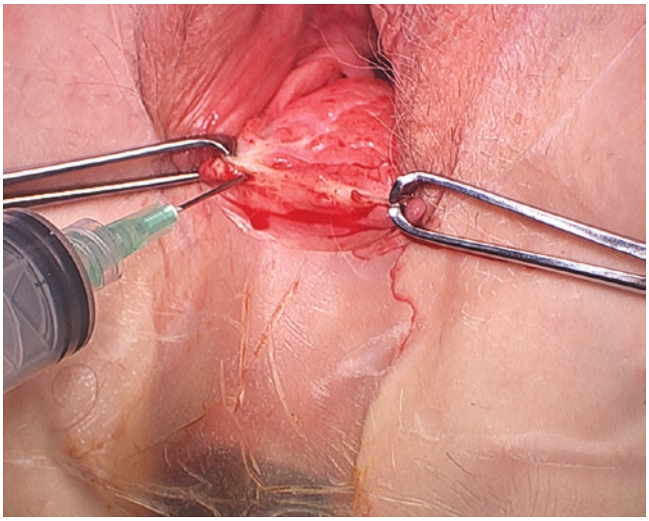
Fig. 15.4 Infiltration of the posterior vaginal wall

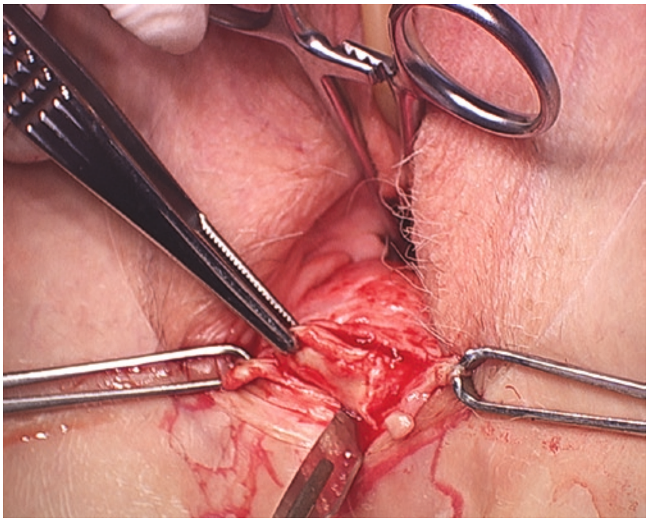
Fig. 15.5 Excision of a triangular piece of skin

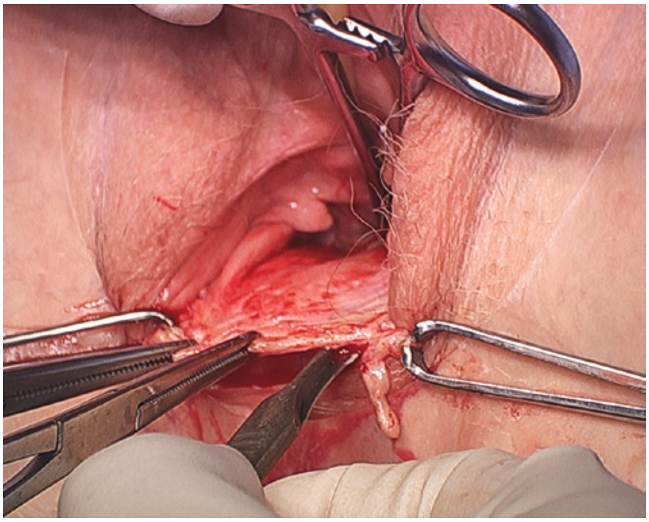
Fig. 15.6 Exposing the transverse perineal muscle

A longitudinal incision is made with scissors along the posterior wall, up to the cervix (Fig. 15.7), exposing the rectovaginal space. The vaginal skin is then dissected laterally on both sides. The presence of pararectal fat is an indication that dissection is near the rectum and pararectal pillar (Fig. 15.8). The index finger is then introduced through the pararectal fat to the right ischial spine, which is exposed by blunt finger dissection, creating a window between the recto vaginal space and ischial spine through the right rectovaginal fascia. The opening is enlarged by inserting the index and middle finger until the sacrospinous ligament is exposed. If there is a large enterocele sac which interfere with the dissection, this will be opened and closed with a buttressing suture using No. 1 Vicryl as high as possible and the sac excised.

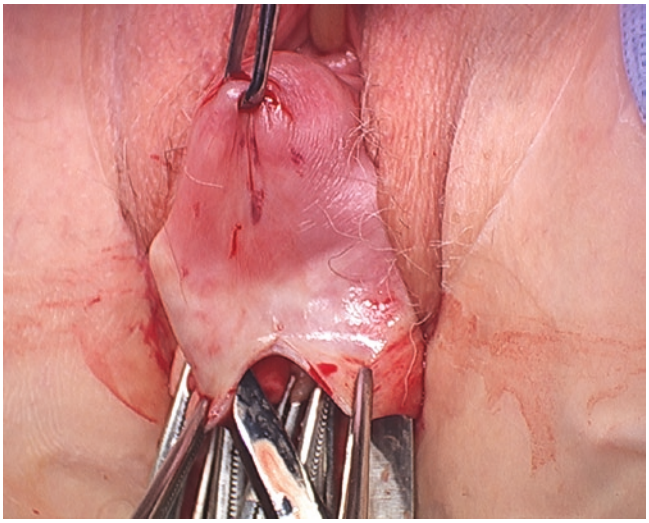

Fig. 15.7 A longitudinal incision is made with scissors along the posterior wall, up to the cervix

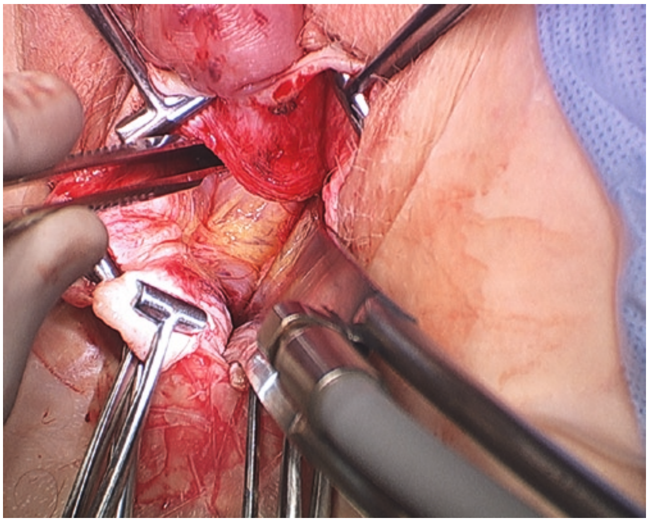

Fig. 15.8 The vaginal skin is dissected laterally on both sides and pararectal fat is exposed

A Miya hook ligature carrier loaded with No. 1 PDS (Polydioxanone monofilament, absorbable suture, Ethicon, UK) (Fig. 15.9) is introduced pointing downward between the right index and middle fingers with the index finger adjacent to the ischial spine. Therefore, a distance of 2 cm medial to the ischial spine is guaranteed. The hook is opened and inserted at or just below the superior margin of the ligament; this step is achieved by sliding the hook up and down between the two fingers and palpating the distinct superior margin of the ligament with the middle finger (Fig. 15.10). The insertion is completed by simultaneous downward pressure on the hook hump with the index finger, and traction on the rear handle of the hook. Penetration up through the ligament is affected by closure and elevation of the handle, a firm bite of the ligament is taken and then the tip of the Miya hook is exposed by pushing the ligament down with the two fingers. A large bite should be avoided because it is unnecessary and will make exposure of the hook's tip rather difficult.

While the assistant holds the elevated handle of the hook, a notched vaginal retractor is inserted using the right index finger to guide it by palpation underneath the hook point. The notch is designed to hook the tip of Miya hook, so it can be visualized and to facilitate the retrieval of the suture.

A lateral pelvic retractor (e.g., Breisky-Navratil retractor) is used to retract the rectum. A loop of the PDS suture is retrieved with a nerve hook (Fig. 15.11). The Miya hook is removed by lowering the handle and guided with two fingers.

The procedure may be repeated so that two sutures are placed into the ligament or two sutures may be inserted at the same maneuver.

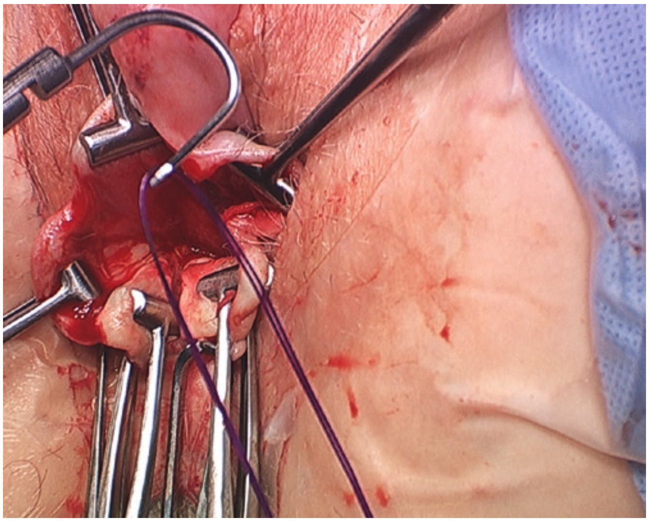

Fig. 15.9 The Miya hook ligature carrier loaded with No. 1 PDS

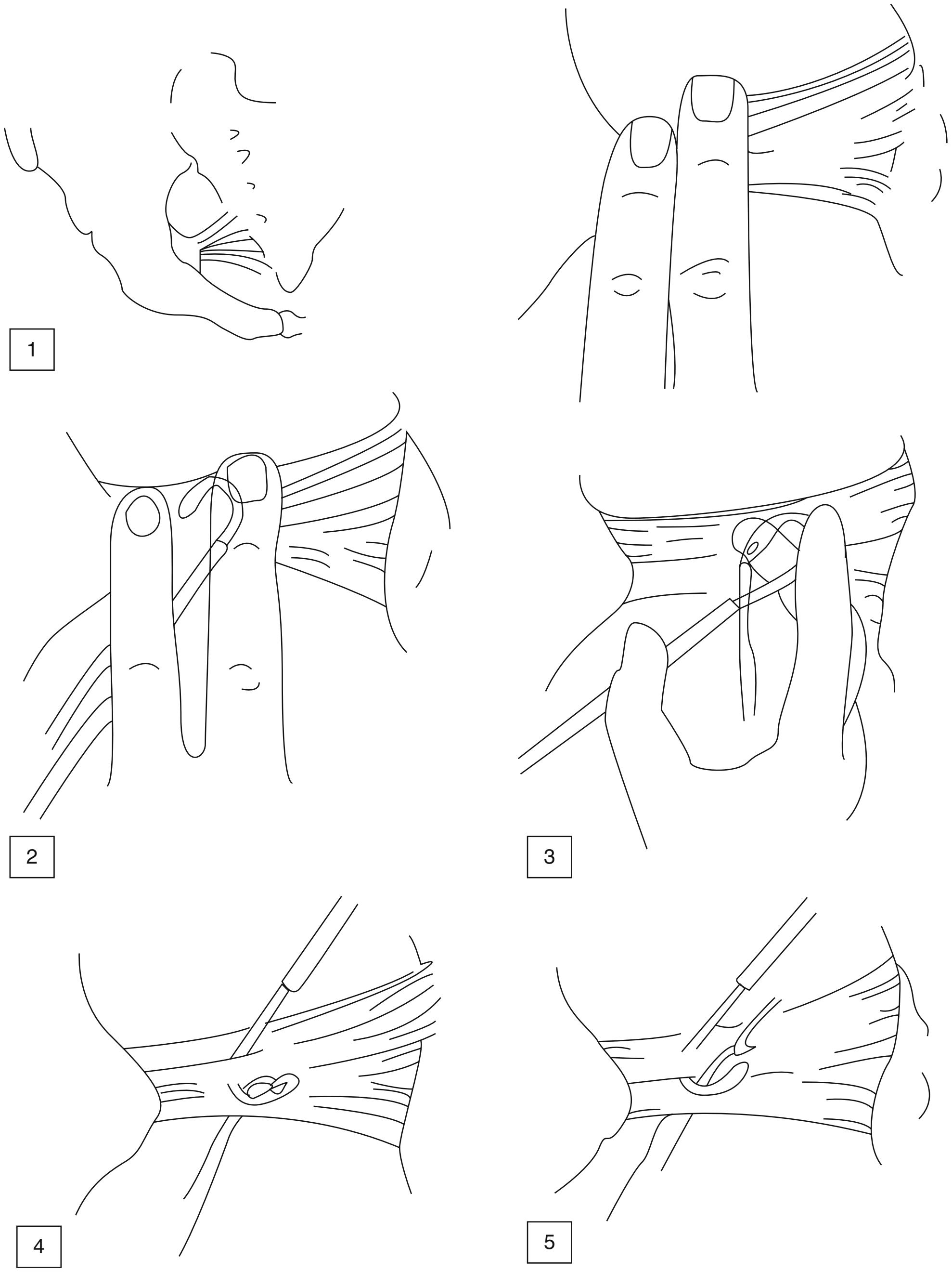

Fig. 15.10 Steps of the insertion of Miya hook and retrieval of the suture (From Hefni[14])

Pulley sutures are created: (Fig. 15.12a–c) The PDS sutures are loaded on to a No.4 Mayo needle and passed through the uterosacral ligament at its cervical attachment and through the adjoining cervical tissue and vaginal skin one on each side of the midline; pulley sutures are inserted on each side of midline to ensure good contact between the cervix and the sacrospinous ligament, when the PDS suture is tied.

The vagina is then closed with a continuous suture with No. 0 Vicryl. The PDS sutures are tied, pushing down with the pulley mechanism, taking the cervix on to the sacrospinous ligament (Fig. 15.13).

The transverse perineal muscle is then approximated (Fig. 15.14a–b) and perineorrhaphy completed. With reasonable accuracy the perineal body accounts for the lower 3 cm of the posterior vaginal wall. The transverse fibers of the deep transverse perinei muscle are the largest component of the perineal body, so the importance of perineorrhaphy cannot be overemphasized.

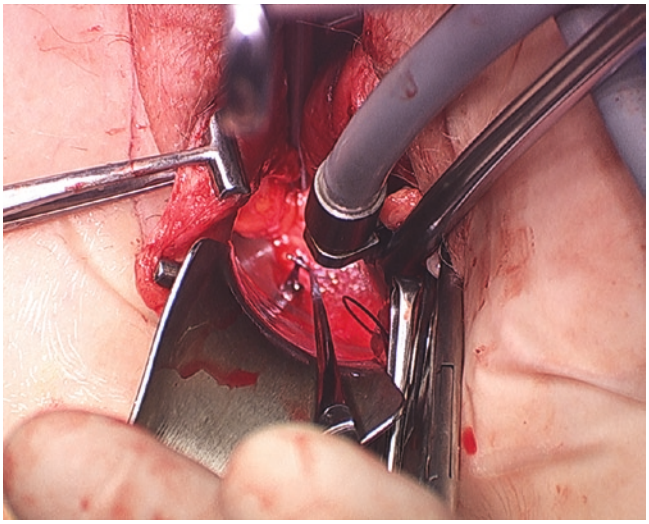

Fig. 15.11 A loop of the PDS suture is retrieved with a nerve hook

Advantages of SSF with Uterine Conservation

A number of studies exist showing the value of uterine preservation at the time of level I genital prolapse repair using sacrospinous fixation (SSF). Maher and colleagues[25] retrospectively compared 34 women after SSF and 36 women after vaginal hysterectomy and sacrospinous colpopexy for symptomatic uterine prolapse. The follow-up period varied between 33 months in the SSF group and 26 months in the hysterectomy group. The two groups were comparable with regard to baseline characteristics including age, parity, body mass index, menopausal status, and degree of level I prolapse. The subjective success rate (defined in the study as no awareness of prolapse, 78% vs 86%, $p = 0.7$), objective cure rate (defined in the study as absence of prolapse beyond the mid-vaginal point, 74% vs 72%, $p = 1.0$) and patient satisfaction (85% vs 86%, $p = 1.0$) were comparable in the two groups, respectively. Moreover, the operating time and intraoperative blood loss were significantly reduced in the SSF group ($p = <0.001$).

Van Brummen and colleagues[26] used a postal questionnaire to compare the outcome of 54 women after SSF and 49 women after vaginal hysterectomy. In the hysterectomy group, the uterosacral and cardinal ligaments were approximated and reattached to the vaginal vault after removal of the uterus to achieve vault support. All operations were performed because of uterine prolapse reaching to or beyond the hymen. Of the 103 women contacted, 74 (72%) returned a completed questionnaire; 44 in the SSF group and 30 in the hysterectomy group. The women in the SSF group recovered more quickly after surgery (odds ratio (OR) = 2.8, 95% confidence interval (CI) 1.1–7.3, $p = 0.04$). No difference in anatomical outcome and recurrence rate between the two techniques was observed.[26]

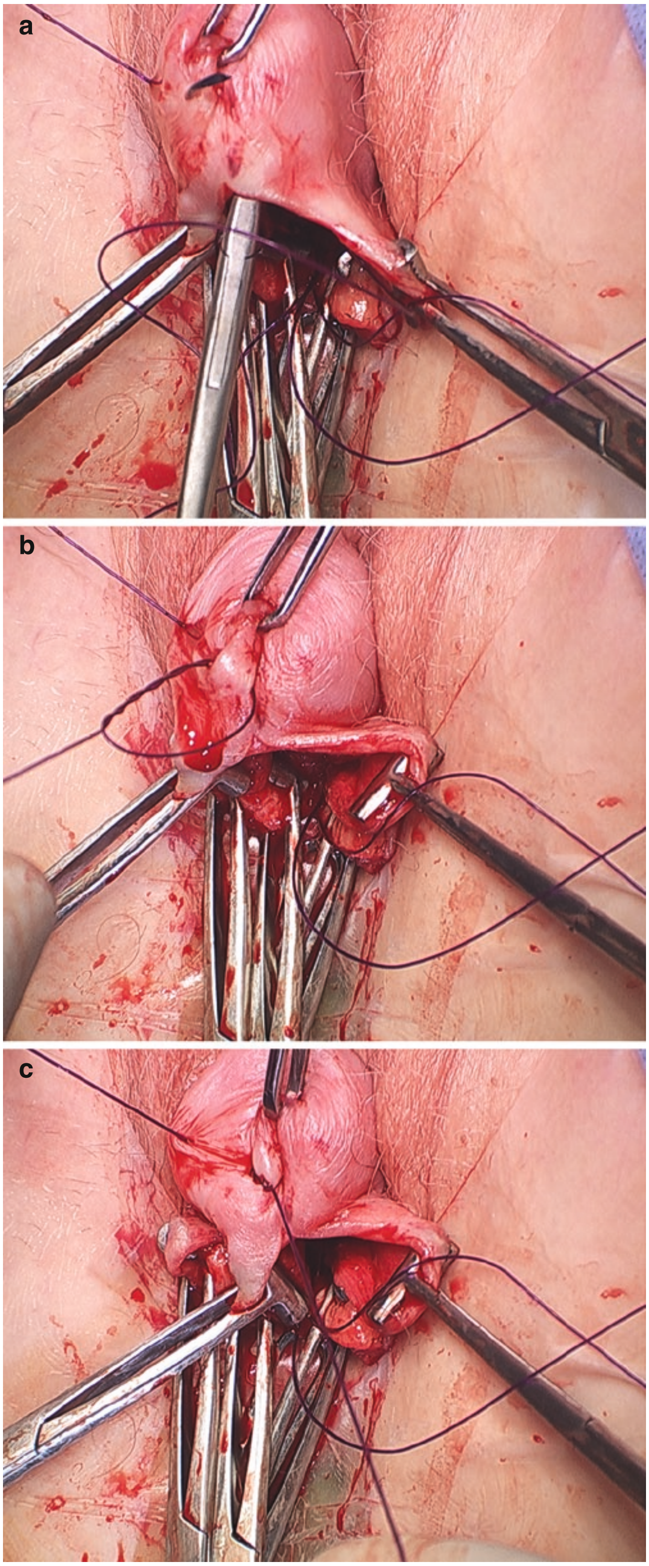

Fig. 15.12 (**a–c**) Pulley sutures are created

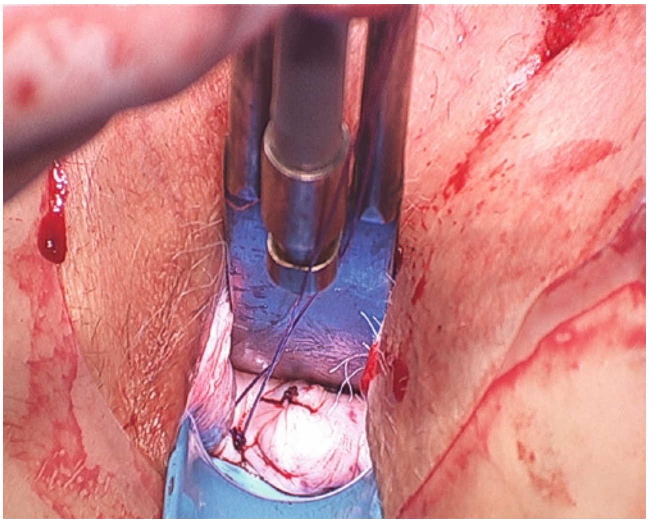

Fig. 15.13 When the PDS sutures are tied, pushing down with the pulley mechanism, taking the cervix on to the sacrospinous ligament

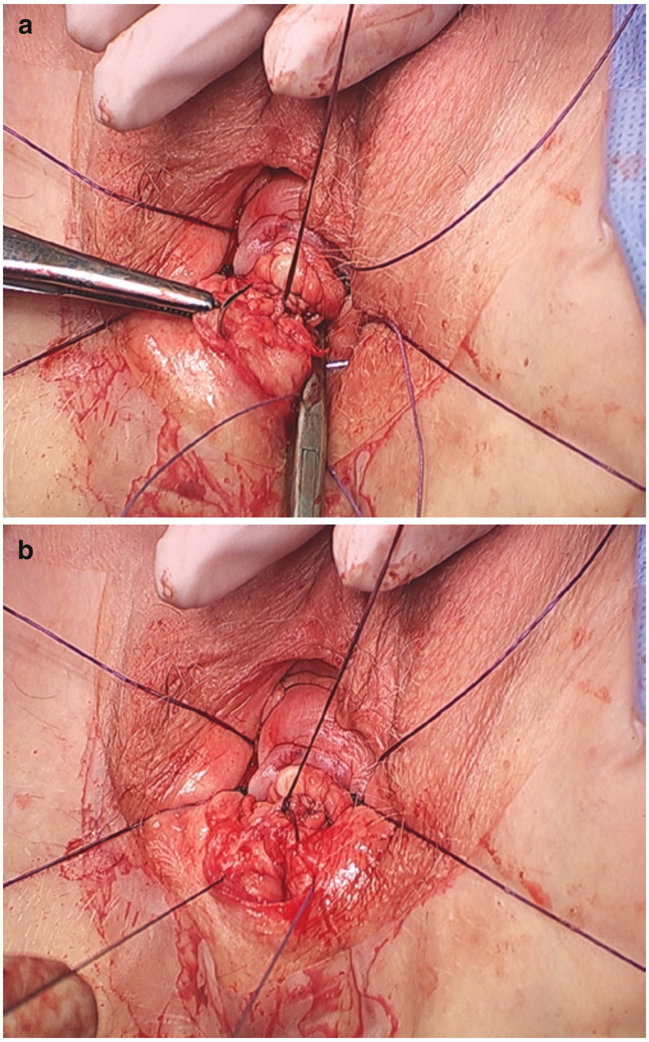

Fig. 15.14 (**a, b**) Approximation of transverse perineal muscle

However, after adjusting for age at the time of surgery, body mass index and length of follow-up using logistic regression, the OR for urge incontinence was 3.4 (95% CI 1.0–12.3, $p = 0.05$) and for overactive bladder was 2.9 (95% CI 0.5–16.9, $p > 0.05$) greater after vaginal hysterectomy, further supporting the beneficial role of uterine conservation at the time of genital prolapse surgery for level 1 defects.

In a prospective controlled study, Hefni and colleagues[21] evaluated the efficacy of sacrospinous cervico-colpopexy with uterine conservation in the treatment of uterovaginal prolapse in 109 women above the age of 60 years with a complaint of symptomatic uterovaginal prolapse. Sixty-one women were treated with sacrospinous cervico-colpopexy with uterine conservation and 48 with vaginal hysterectomy and sacrospinous colpopexy. The mean age for the two groups was comparable (70.1 years vs 69.4 years, respectively; $p = 0.8$). Women who had uterine conservation had significantly less operative blood loss ($p < 0.01$), shorter operating time ($p < 0.01$), and fewer complications after surgery ($p = 0.01$) compared with the hysterectomy group. After a mean follow-up duration of 33 and 34 months, respectively, the two groups had comparable success rates with regard to uterine and upper vaginal support (93.5% vs 95.9%, respectively; $p = 0.6$). During follow-up, three patients (5%) in uterine conservation group and two patients (4.2%) in hysterectomy group underwent repeat operation for recurrent uterovaginal or vault prolapse.

More recently, Dietz and colleagues[27] followed up 99 women for a mean duration of 22.5 months after SSF for symptomatic uterovaginal prolapse. Recurrent uterine prolapse requiring surgery occurred in 2.3% only of women. Moreover, 84% of women were highly satisfied with their outcome and 91% would recommend the procedure to a friend. The same group[28] assessed the functional outcome after sacrospinous hysteropexy performed for 72 women with symptomatic uterovaginal prolapse using a standardized and validated questionnaire. The study results showed that all urogenital symptoms including those of urinary incontinence, overactive bladder, and obstructive micturition and several defecatory symptoms such as constipation and obstructive defecation were reduced, and the quality of life domains were improved after surgery. In addition, sacrospinous hysteropexy anatomically cured the uterine prolapse in 93% of the 72 women. Likewise, numerous studies have demonstrated that sexual function is maintained in sexually active women after sacrospinous hysteropexy for uterovaginal prolapse.[29-34]

Why Is Hysterectomy Unnecessary in the Treatment of Uterine Prolapse?

As demonstrated above, Level 1 is represented by the parametrial ligaments, which continue down the sides of the upper vagina as the paracolpium. Damage to this level of support will lead to apical (i.e., uterine and upper vaginal) prolapse. *The uterus itself plays a passive role in this process*[35,36] *and its removal does not address the underlying pelvic organ support weakness or improve the outcome of the repair procedure.*[21,25,37,38]

Our study of 120 SSF with uterine preservation shows that SSF with conservation of the uterus is associated with a high long-term success rate. The objective cure rate in the study was 91%, which is comparable to that reported by various researchers after sacrospinous vault fixation performed at the time of hysterectomy.[36,39,40]

Uterine preservation has many advantages. Operative morbidity and hospital stay are reduced compared to when hysterectomy is performed[2,20,21,25,41]. This is particularly relevant in older women in whom it is critical to minimize operative morbidity.[21,42] Lambrou and colleagues[43] reviewed the prevalence of perioperative complications among women undergoing pelvic reconstructive surgery and found that operative complications were strongly associated with the number of surgical procedures performed, operating time, and blood loss. Therefore, additional surgery that has little to add to the outcome of genital prolapse repair should be avoided.

The advantages of uterine preservation extend beyond the early postoperative period. In addition to positive psychological impact related to body image and self-confidence[44,45], there is evidence that uterine preservation is associated with a reduced risk of urinary dysfunction[26,46-50]. This could be explained by avoiding bladder dissection or division of the pericervical ring of connective tissue, both of which are inevitable during hysterectomy.[47,51] In our study, we observed a significant reduction in the prevalence of urinary symptoms after surgery ($p < 0.01$). There was also a 40% reduction in bowel symptoms after surgery, although this did not reach statistical significance because of the small number of patients who presented with bowel symptoms in our study (14%).

More recently, it has become apparent that preserving the uterus during pelvic reconstructive surgery, which involves the use of synthetic mesh, is associated with a lower risk of mesh erosion compared to when hysterectomy is performed.[52,53] Given the rapid rise in recent years in the number of pelvic repair and incontinence procedures that involve the use of synthetic material[3], the scope for uterine preservation in prolapse surgery is likely to expand.

Reproductive Function and Pregnancy After SSF and Uterine Conservation in Young Women

Uterine preservation maintains reproductive function in younger women wishing to preserve fertility. A national survey in the USA showed that 18% of prolapse procedures were performed in women aged 20–40 years.[54] With the current trend toward delaying motherhood till later in life, the demand for uterine preservation is likely to grow.

From a reproductive perspective, SSF is superior to other uterus-sparing repair procedures as it is an extraperitoneal operation and involves no trauma to the cervix. Thus, it avoids potential compromise to tubal or cervical function associated with intraperitoneal[20,24] operations.

Pregnancy after SSF with uterine preservation is possible.[30] Between 1994 and 2004, we performed 120 SSF with uterine preservation, including 10 women of reproductive age. During follow-up, three of these ten women (mean age 26 years, range 22–30; parity 0–1) conceived naturally and were delivered at term by elective Cesarean section to avoid the 20–50% risk of prolapse recurrence after vaginal delivery reported previously[25,31]. All three women were reviewed at least 2 years (mean 3.3, range 2–5 years) after delivery and no evidence of recurrent uterine prolapse was seen. Although performing elective Cesarean section at term represents an appropriate management, the available data regarding the optimum mode of delivery after uterovaginal prolapse repair and uterine preservation is limited to fewer than 20 cases.[25,31-34] A larger number of patients and long post-delivery follow-up duration are needed before a recommendation regarding mode of delivery could be made.

As women opting for uterine conservation are generally younger and sexually active, it is reassuring that the vast majority (95%) of sexually active women in our study reported either no deterioration or improvement in sexual function during follow-up. This is in line with previous studies[55,56], which reported a positive overall effect of sacrospinous fixation on sexual function. In addition, SSF maintains vaginal length and capacity for sexual intercourse.[57]

Is Hysterectomy Feasible After Conservative Repair?

There are two issues that are often raised following SSF with conservation of the uterus for uterine prolapse. One is the ease of obtaining a cervical smear or endometrial biopsy following the surgery and the other is feasibility of doing hysterectomy at a later stage if that becomes necessary.

We encountered no difficulties in taking cervical smear or performing hysteroscopy or hysterectomy following the SSF, since the cervix returns to its normal position 6 weeks following surgery, even when the SSF is performed on one side – usually using the right sacrospinous ligament.

In our study of 120 women with symptomatic uterovaginal prolapse who were treated with sacrospinous cervicocolpopexy as part of their pelvic floor repair between September 1994 and December 2004, eight women underwent vaginal hysterectomy within 3 years of SSF because of recurrent uterovaginal prolapse, while one had abdominal hysterectomy and bilateral salpingo-oophorectomy for benign bilateral ovarian thecoma discovered 5 years after SSF. No complications occurred during any of these procedures.

In our experience, gynecological surgery after SSF is uncomplicated. Endometrial surveillance, vaginal hysterectomy for recurrent prolapse and abdominal hysterectomy for non-prolapse-related pathology have been performed without difficulty. This is in accordance with the study of Maher and colleagues[25], which reported no complications encountered during two vaginal and one abdominal hysterectomy performed after sacrospinous hysteropexy.

Surgical Complications and Management

Major complications are rather rare but could be serious, so it is important to be aware of these complications and how to manage them.

Bleeding

Major bleeding can occur from one of the three sites: the pudendal vessels, the inferior gluteal vessels, or the rectal venous plexus. The avoidance of bleeding complications is simple. Never insert the Miya hook ligature carrier lateral to the ischial spine, above the superior margin of the ligament, or too medial in the ligament toward the sacrum. If the bleeding is from the venous plexus, it usually settles down and leaving a drain may be advised. In case of severe bleeding from the pudendal vessels or inferior gluteal vessels, it is rather difficult to control by suturing or clamping, so it may be safer to use pressure to stop the bleeding by inflating a catheter balloon containing 50 or 100 mL of fluid and leaving it for 24–48 h before deflating the balloon and removing it. Arterial embolization has also been suggested.[58]

Nerve Injuries

The major nerves running in this area are the pudendal nerve, and the sciatic nerve and its branches, which are just above the superior margin of the ligament. Once again, to avoid injury to these nerves, insertion of the Miya hook must be medial to the ischial spine and at or below the superior margin of the ligament. If any of these injuries occur, removal of the suture is advisable.

Buttock pain after sacrospinous colpopexy has been described and is not uncommon. Its incidence in our patient population is about 6%. The pain is caused by deep insertion of the SSF suture because the perforating cutaneous nerve, which usually arises from the posterior aspect of the second and third sacral nerve, runs between the sacrospinous and sacrotuberous ligament, winding around the inferior border of the gluteus maximus and supplying the skin covering the medial and lower part of that muscle.

Buttock pain is usually temporary lasting for less than 2 weeks in the majority of cases with no need to remove the suture. However, in rare instances it could last up to a maximum of 6 months.[14]

Another nerve that could be injured during SSF is the posterior femoral cutaneous nerve, which arises from the ventral division of the second and third sacral nerves, and usually runs between the sacrospinous and sacrotuberous ligaments. This nerve supplies the skin of the back of the thigh down to the back of the knee and, if it is caught in the suture, loss of sensation and numbness may result in this area. Once again, avoidance of deep suture placement in the sacrospinous ligament will prevent this complication.

There is evidence that nerves are present and widely distributed within the body of the sacrospinous ligament.[59] A wide variety of sizes and nerve thickness have also been demonstrated, suggesting a variety of functions, including possible pain reception. This fact should be taken into consideration when planning the fixation of the vagina into the sacrospinous ligament.

Injury to Bowels, Rectum, and Soft Tissue

Injury to the bowels could happen if the enterocele sac is missed during the dissection and contained a loop of bowel. Injury to the rectum could occur as a result of the sharpness of the Miya hook end; therefore, protection of the Miya hook between insertion and removal is essential. Also, using several large retractors is not only unnecessary but will cause soft tissue damage. Only one retractor is usually required; a lateral pelvic retractor, such as Simon's retractor or the Breisky–Navratil retractor, may be used to retract the rectum toward the medial side during retrieval of the suture.

Damage to the Vaginal Wall

Particularly in postmenopausal woman, the vagina could be quite thin and atrophic, and could be torn during the last step of the operation, tying the PDS suture. To avoid this, preoperative preparation of the vagina is quite important. If the vagina is atrophic and thin, estrogen cream should be used for 2–3 weeks before surgery. One of the main reasons for failure of this surgery is the presence of a short vagina. To overcome this problem, a vaginal dilator with vaginal estrogen cream should be used for several weeks before admission. This will provide some additional length to the vagina to enable the surgeon to approximate it reliably to the sacrospinous ligament.

Conclusion

The uterus itself plays a passive role in the process of uterine prolapse and its removal does not address the underlying pelvic organ support weakness or improve the outcome of the repair procedure.

Uterine conservation at the time of SSF offers distinct advantages while correcting level I uterovaginal prolapse. It is associated with reduced operative and postoperative morbidity and a lower risk of mesh erosion compared to when hysterectomy is performed. It also maintains reproductive activity in younger patients and avoids potential compromise to tubal or cervical function associated with intraperitoneal prolapse repair procedures.

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Uterine Prolapse Repair with Meshes

16

Peter von Theobald

Introduction

Beginning in the late 1980s, we have seen, gynecological surgery turning the page of a new era. The first sign was the revolution of the laparoscopic surgery that shifted from nonexistent to golden standard within 10 years. Before this boom, the rule was the dogma: the trainee reproduced his master's techniques as the master himself applied his own master's procedures. There were different and very exclusive schools, each believing that only they knew the truth and that others had to be convinced of their ignorance. Those who tried to change techniques were considered heretical.

The opening of most national borders, the internet, new scientific knowledge, and technological advances contributed to the emergence of a new dogma: that there is no truth, no certitude; the era of exchange and information. New ideas and techniques spread very quickly. The global trend toward less and less invasive and more and more ambulatory surgery encouraged surgeons to develop new concepts. Laparoscopy was one of these new concepts, just as mesh surgery is now.

What is a mesh? First it is a foreign body implanted in a selected place, where collagen tissue is weak. It provokes an inflammatory reaction, attracts macrophages and other inflammation cells, and, finally fibroblasts that will produce collagen fibrosis around this foreign body. As long as the foreign body stays in place, this collagen tissue will be maintained and renewed. Thus, by implanting a nonabsorbable mesh, we oblige the patient's body to repair itself with autologous collagen.

What is mesh not? It is not a mechanical support or suspension of the pelvic floor. Its aim is to restore the correct axes of the vagina. This requires a good knowledge of the functional anatomy, skilful dissection, and repair. There is no need for big forces or very strong mesh resistance to tearing. Even the weakest mesh is stronger than the strongest ligament.

Is mesh surgery new in Urogynecology? No. It has been used in open sacrocolpopexy since more than 50 years (first publication 1958 by Hughier and Scali).[1] Long series have shown the excellent tolerance and the long-term effectiveness of these abdominal procedures. Erosion and infection rates in serious series are around 5%. Many materials have been tried and polypropylene seems to be one of the best tolerated (and probably the cheapest). But no correct comparative clinical trials have been led to date and probably never will because the expected difference of erosion or infection rate appears to be underneath the 5% barrier and thus, hardly may reach statistical significance.

The new concept is the use of prostheses in vaginal prolapse surgery, priory prohibited, recognized feasible since 1997: since the boom of the suburethral vaginal slings.

Adequate treatment of genital prolapse requires a defect-specific approach. Repair of upper compartment prolapse, called level 1 (vaginal vault, uterine prolapse, enterocele) can be performed with abdominal or laparoscopic techniques such as sacrocolpopexy (SCP)[2–9]; the Kapandji-type operation[10,11]; combined abdominal/vaginal techniques[6,11,12]; or techniques using the vaginal route such as fixation to the sacrospinous ligament (SSLF),[13–16] MacCall-type culdoplasty,[17] or the traditional Manchester operation. Peter Petros[18] described a new technique using a sling of polypropylene mesh for suspension of upper compartment organs which have prolapsed, called posterior intra-vaginal slingplasty (PIVS), and for which a more detailed name would be "infracoccygeal translevatorial colpopexy." Advantages of PIVS are the following:

- No need for laparotomy as for abdominal SCP or Kapandji operation
- Shorter learning curve than laparoscopic SCP or Kapandji operation
- Much less dissection required as for the other vaginal procedures (SSLF, Manchester and MacCall); Thus, reduction of the postoperative pain
- No lateral deviation of the vagina as for SSLF
- No risk of ureteral injury as for MacCall procedure
- Much less tension and thus again, much less postoperative pain than in any other vaginal procedures

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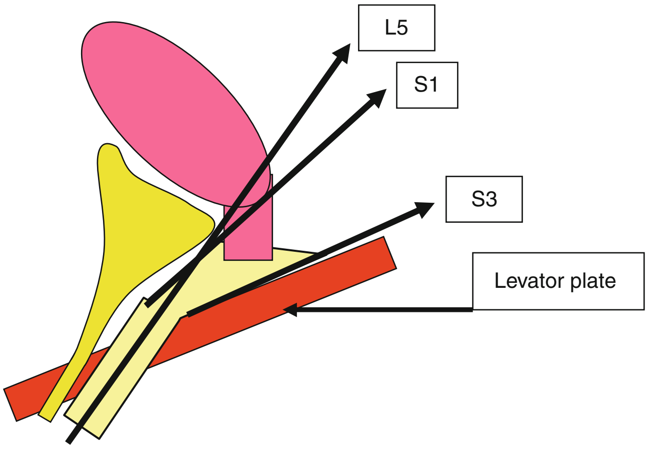

Fig. 16.1 The different axes of the vagina

The concept of the PIVS is to reconstruct a level 1 support by pulling the vaginal vault and the uterine cervix toward the sacral concavity in direction of the third sacral vertebra. Applying the posterior vaginal wall on the levator plate and restoring the double axis of the vagina that is the key point of pelvic floor stability (Fig. 16.1).

As developed in the previous chapter, hysterectomy is not the treatment for prolapsed uterus. Unless there is a specific uterine disease requiring hysterectomy (symptomatic myomas, endometrial or cervical diseases, very bulky hypertrophy of the cervix), conservative surgery will avoid useless per operative bleeding, postoperative pain, and longer hospital stay. PIVS is a very effective procedure to restore normal position of the cervicovaginal complex. Compared to the modified SSLF (with uterine conservation), it keeps the normal central position of the cervix and creates a "neo ligament" around the tape when SSLF relies on the hold of a single unilateral suture. Much less tension is involved with the PIVS because it does not require a contact between the sacrospinous ligament and the uterine cervix. Compared to the Manchester procedure, it does not need that huge uterosacral ligament dissection. The "neo ligament" created around the tape provides a much stronger hold than the suturing of a weak uterosacral ligament.

Surgical Technique

Insertion of the PIVS tape, and treatment of any existing rectocele requires standard posterior sagittal midline full thickness colpotomy, without opening the perineum (if it can be avoided) in order to keep pain to a minimum. The rectovaginal fascia is left on the mucosa. The top of the incision is at 1 or 2 cm from the cervix of the uterus. The rectovaginal plane and enterocele pouch are dissected. The two para-rectal fossas are opened using the finger and blunt-tipped scissors. The landmarks at each side are the ischial spine, the sacrospinous ligament and the levator ani muscles (iliococcygeous and coccygeous muscles). Upward, the uterine isthmus and its junction with the uterosacral ligaments are visible. This dissection is carried out without any retractors. A 5 mm incision is made 3 cm lateral and inferior to the anal verge on each side. The IVS 02 Tunneller® (Covidien, USA) is inserted via this buttock incision into the ischiorectal fossa, separated from the rectum by the levator ani muscles and the surgeon's finger which is inserted via the para-rectal fossa. This finger keeps a check on the movements of the tunneller through the muscle layers. The blunt tip of the tunneller is maneuvered to a position where it is in contact with the sacrospinous ligament, and 2 cm medial to the ischial spine. The coccygeous muscle, overlying the sacrospinous ligament, is then perforated at this level by the blunt tip that comes into contact with the surgeon's finger. Thus covered and protected from any contact with the rectum, the blunt tip of the tunneller is taken out of the colpotomy area. The IVS® polypropylene tape is pulled through the tunneller using the plastic stylet, and then the tunneller is removed. The tape is fixed to the uterosacral ligaments and the uterine isthmus using two absorbable sutures. If there is a concomitant rectocele, a pre-shaped rectovaginal interposition prosthesis measuring 8 cm long and 4 cm wide with two arms (Parietene Duo®, Covidien, USA) is used. The aim is to cover and reinforce the rectovaginal septum in order to correct the rectocele. The bottom is sutured to the central fibrous core of the perineum on each side of the anus, again using two stitches of absorbable suture. The prosthesis must lie flat against the rectum, with no large creases and pulled up into the sacral concavity at the same time as the vaginal vault and the uterus. No colpectomy is used here either. The posterior colpotomy is closed with rapid absorption suture prior to pulling on the two external ends of the PIVS mesh. A vaginal pack is inserted into the vagina for 24 h in order to ensure that the vaginal walls are properly in contact with the prostheses and the dissection planes. A bladder catheter is inserted for the same period of time (Figs. 16.2–16.5).[19]

Our Series

We published a prospective, observational study[20] of 108 consecutive patients, with a mean age of 60 years (range 36–82). Patients presented with genital prolapse giving rise to symptoms, were included in our Department of pelvic floor surgery between August 2001 and July 2003. To be eligible for inclusion, the prolapse had to include descent of upper compartment organs (vaginal vault, hysterocoele, enterocele) with a point $C > 0$ cm according to the POP-Q classification.

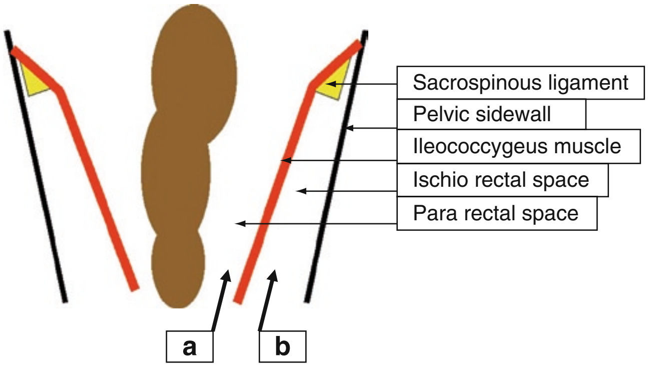

Fig. 16.2 Frontal section of the pelvis: the surgeons finger (**a**) is in the para-rectal space, guiding the tunneller that is in the ischiorectal space (**b**)

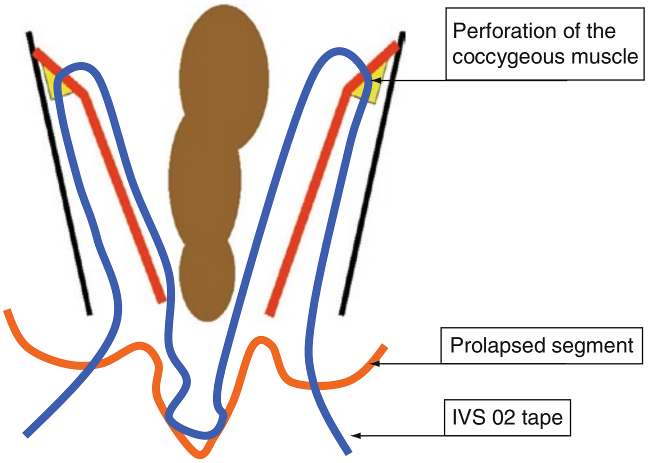

Fig. 16.3 Frontal section of the pelvis: the perforation of the coccygeous muscle at the level of the sacrospinous ligament is the pulley that will raise the prolapsed segment

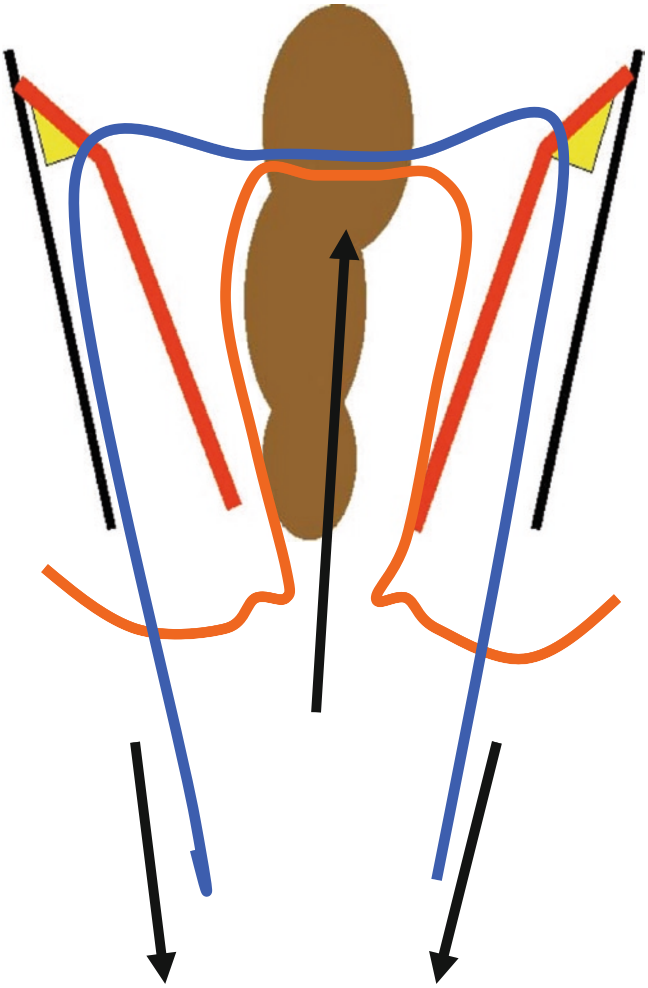

Fig. 16.4 Frontal section of the pelvis: suspension of the prolapsed segment

Cystocele and/or rectocele, if associated, were given specific treatment. All patients underwent PIVS, and in addition, those with an associated cystocele or a rectocele were treated with placement of a polypropylene mesh in the vesicovaginal or rectovaginal space respectfully. Hysterectomy was not performed to treat prolapse. Rather, hysterectomy was only performed for medical indications such as meno- or metrorrhagia with a polymyomatous uterus, symptomatic uterine hyperplasia, or cervical dystrophy. In case of isolated hypertrophic lengthening of the cervix, trachelectomy was carried out. When stress urinary incontinence was diagnosed at clinical examination with full bladder or when the closing pressure was less than 25 cm water, a suburethral tape was inserted using the anterior intravaginal slingpplasty (IVS) technique via a separate vaginal incision beneath the mid-urethra. All patients were seen 6 weeks postoperation, again after 6 months and then every year by the surgeon or another gynecologist in the department. The main study criteria were patient morbidity (perioperatively and immediately postoperatively, as well as long-term morbidity) and also the anatomical and functional results at short term with respect to the PIVS. The secondary study criteria were the same with respect to the insertion of vesicovaginal and rectovaginal interposition prostheses.

The PIVS operation was performed as planned in all 108 cases. Thirty three patients had a past history of hysterectomy or surgery for prolapse of the upper or posterior compartment. We performed 19 hysterectomies for miscellaneous medical reasons. This leaves a subgroup of 56 patients in this series who had a conservative uterine prolapse repair. We performed 22 amputations of the cervix (39%). From a functional point of view, all the patients had previously complained of a dragging sensation in the pelvis and the uncomfortable presence of a protruding mass. Twenty seven patients had also complained of stress urinary incontinence, ten of stubborn constipation that worsened concomitant with the prolapse, two of anal pain at defecation and one of anal incontinence.

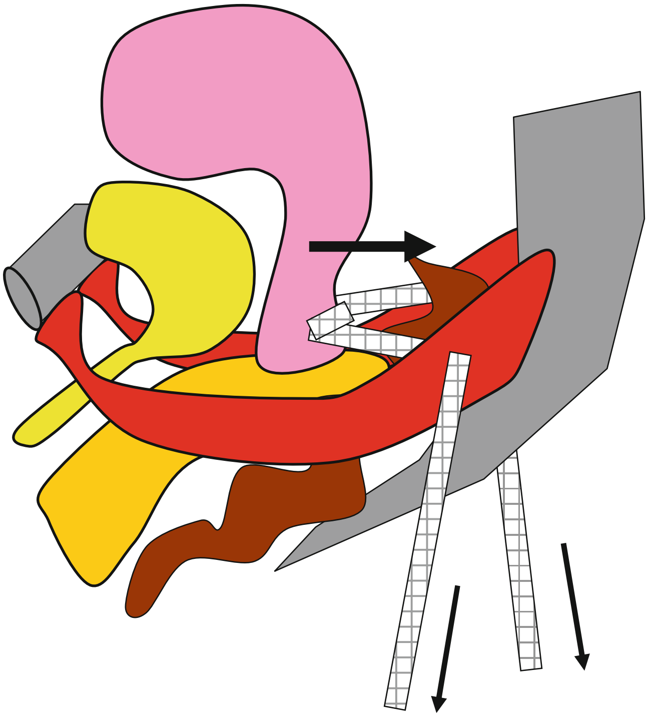

Fig. 16.5 Three-dimensional vision of the PIVS attached to the uterine cervix, pulling toward S3 and repositioning the posterior vaginal wall on the levator plate

Concerning these 56 patients, the intraoperative complications (nine cases) were essentially two bladder injuries: one during dissection of the cystocele and one during the passage of the suburethral sling insertion device. The postoperative complications consisted of anemia (loss of more than 2 g/dL of hemoglobin) in three cases (4.8%). With respect to the cystocele correction, one vaginal erosion occurred at 2 months that was resolved by simple excision of the exposed mesh under local anesthesia. For the treatment of the upper and posterior compartments, there were two infections of the prosthetic materiel which had to be ablated completely, with one case occurring on a hematoma of the para-rectal fossa (on day 15) and the other on a vaginal erosion at 5 months. Finally, there were three with postoperative urinary infection and one case of isolated fever, which resolved without complications. The average hospital stay was 2.9 days (ranging from 2 to 6 days). No immediate reoperation was necessary. The mean follow-up of the patients who were seen again was 19 months (ranging from 9 to 31 months). Two patients were lost to follow-up. They had no intraoperative complication and their characteristics (age, past history, type of operation) were similar to those of the total cohort.

From an anatomical perspective, the presence of a prolapse at the first postoperative consultation at 6 weeks was considered as a failure, whilst if the same was found later, this was considered as a recurrence. With regard to correction of the upper and posterior compartments (assessment of PIVS in 54 patients), there was one failure in the patient whose prosthesis was removed on day 15. There were two recurrences of uterine prolapse at 6 months, one of which occurred in the patient who had an infection on the prosthesis at 5 months with, once again, complete ablation of the mesh.

From a functional point of view (in 54 patients), and with regard to PIVS and the posterior prosthesis, the results included two cases of moderate de novo constipation, one case of dyspareunia that resolved after section of one of the two PIVS side strips, and also two cases of urinary incontinence that were unmasked by the operation. However, in the seven patients who presented with preoperative dyschesia, four had no more symptoms and one had experienced considerable improvement.

In literature, several series have been published. De Tayrac[21] has published a randomized trial versus SSLF; PIVS is quicker and easier to perform, post-operative pain is reduced ($p < 0.01$). Cure rates, quality of life and sexuality questionnaires (PFIQ, PISQ), and symptoms scores (PFDI) are similar after 17 months of follow-up. Postoperative cystocele occurred in 25% of SSLF and 4.8% of PIVS ($p < 0.05$). In a recent retrospective analysis of 87 patients with 27 months of follow-up, the same author[22] showed some superiority of the monofilament tapes compared to the multifilament ones: 9% extrusion versus 0%. But no difference in the results: 18% prolapse recurrence rate versus 14% ($p = 0.79$). Hefni[23] has published an observational study of 127 patients with 14 months of follow-up, using the multifilament tape. The upper genital support was maintained in 88% of patients. Tape erosion was higher in patients over 60 (RR = 1.6) and current treatment for diabetes (RR = 4.95). Neuman[24] found similar results on a prospective study on 79 women with uterine descent. 44 underwent hysterectomy upon their own request and 35 wished to keep the uterus. Both groups were treated with the multifilament PIVS. Follow-up was 29.8 months. The global cure rate was 98.7%, satisfaction rate 89.9%, with no significant difference between the two groups. The only difference found was the hospitalization period: 4.2 for the hysterectomy group versus 1.5 in the other one. Erosion rate was 12.7%.

Conclusion

In conclusion, the use of meshes is certainly the future for conservative management of the uterine descent. The meshes are improving quickly: monofilamentar, becoming lighter and having bigger pore size. Indications still have to be discussed between laparoscopic sacrocolpopexy and PIVS.

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Anterior and Posterior Enterocele

17

Carl W. Zimmerman

Enterocele is defined as a hernial protrusion through the vesicovaginal or rectovaginal pouch.[1] Implied in this definition is the presence of a defect or defects in the normal continuity of the endopelvic connective tissue within the area adjacent to the cervix, or, if the cervix is absent, the hysterectomy scar. Enteroceles are considered true hernias.[2] In normal female human pelvic anatomy, the uterine cervix is suspended and supported within the interspinous diameter by a complex set of connective tissue elements known as the endopelvic fascia (see Chap.1). Suspension and circumferential stabilization of the cervix is a function of the uterosacral, cardinal, and pubourethral ligaments as they insert on the pericervical ring. Anteriorly, the pericervical ring receives the pubocervical septum (fascia), and posteriorly the rectovaginal septum (fascia). These septa are stabilized laterally by the arcus tendineus fascia pelvis and prevent descent of the bladder (cystocele) and rectum (rectocele) into the vaginal vault. Lateral disruptions of these septa are known as paravesical or pararectal and paravaginal defects. Anterior and posterior enterocele formation occurs when the apical portion of either septum is disconnected from the pericervical ring. Potential causes of failure of the apical septal attachment may be related to inherently weak tissue or traumatic. Both general causes of failure likely play a role in the majority of cases of enterocele. Currently, a significant amount of research is being done to characterize the role that various types of collagen play in various parts of the body including pelvic organ prolapse. The fact that only 11% of women will eventually develop symptomatic prolapse clearly means that some variation exists among individuals. Aging undoubtedly plays a role as prolapse becomes progressively more common as senescence progresses, and the same can be said with regard to the menopausal state. Therapeutic administration of steroids is known to weaken connective tissues and is believed to play a role in the body's natural metabolic ability to continually remodel all collagen-containing tissues in the body including those in the pelvis. When normal innervation of the pelvic floor is not present, prolapse becomes more common, as in patients with pudendal neuropathy, spinal cord injury, or spina bifida (Table 17.1). The absence of the support of the muscular pelvic floor in this type of circumstance forces the pelvic connective tissues to bear a larger and more constant amount of strain than is normal. This constant load bearing results in failure of the connective tissue over time (Table 17.2).

Connective tissue trauma within the pelvis is almost always a result of the substantial forces of childbirth. Once the connections of the endopelvic connective tissue are weakened or disrupted by parturition, other mechanical factors may serve as secondary etiologic factors in mechanical disruption of theses tissues.[3] Vaginal delivery is considered a primary etiologic factor for pelvic organ prolapse and all other factors are considered secondary in the vast majority of cases.

A detailed analysis of the effect of the descent of a fetal head though the interspinous diameter is critical to the understanding of how pericervical connective tissues are disrupted and the pattern of disruption that can be found.[3] All named components of the endopelvic fascia intersect with the pericervical ring that is within the interspinous diameter in normal anatomy. Certainly, these fascial connections are subjected to a very large amount of stress by the descent of the fetal head. Descent of the fetal cranium

Table 17.1 Etiology of endopelvic connective tissue weakness

Collagen abnormalities
Senescence
Hypoestrogenism
Chronic steroid therapy
Neuropathy

Table 17.2 Etiology of traumatic disruption of endopelvic connective tissue

Childbirth
Lifestyle
Chronic cough
Chronic straining

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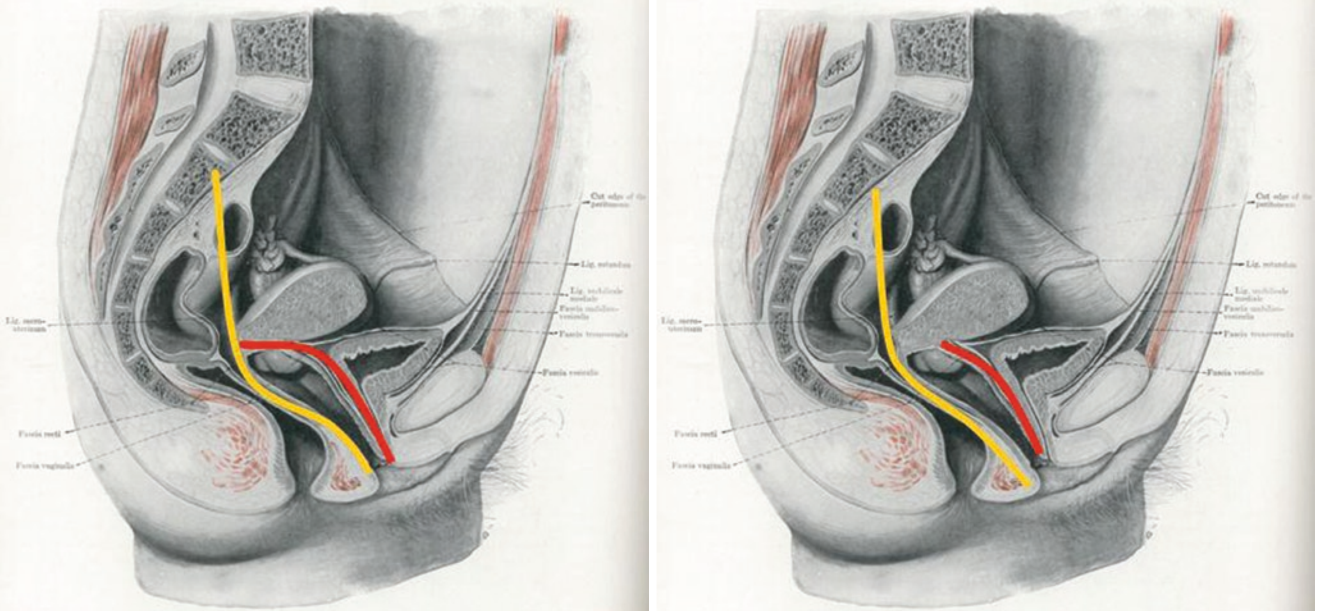
though the interspinous diameter requires several cardinal movements of labor and molding of the head. From a connective tissue standpoint, for normal vaginal delivery to cause prolapse requires disruption of connective tissue connections and their subsequent displacement away from the interspinous diameter. The result is a typical pattern of fascial damage as outlined in Fig. 17.2. Disruptions of the three-dimensional integrated connective tissue continuum known as the endopelvic connective tissue is centered within the interspinous diameter and the specific defects can be attributed to the passage of the fetal cranium through that narrow plane of the pelvis. For example, most disruptions of the pubocervical septum are twofold. Approximately 90% of the time, the paravesical paravaginal defect is on the right side of the patient consistent with a left occipitoanterior pattern of delivery. This mechanism of delivery places the rotating arc of the descending fetal head in the maternal right hemipelvis when the fetal head is above the interspinous diameter and is responsible for a shearing of the pubocervical septum away from the arcus tendineus fascia pelvis in the area adjacent to the ipsilateral ischial spine. This defect is called a paravaginal defect and is important in the formation of anterior enterocele. The other pubocervical defect is an apical transverse disruption of the fascia at its junction with the anterior cervix and is a result of the same pressure that creates complete cervical dilation.[3] Once the pubocervical septum is disrupted on two contiguous sides, anterior vaginal prolapse can develop because the mechanical stability of the septum is compromised. Notice that the old concept of cystocele due to a generalized attenuation of the pubocervical septum[4] is not valid as anything more than a secondary concept in this line of reasoning. Site-specific defects explain all of the disruptions that are necessary for development of anterior enterocele. In fact, anterior enterocele and central cystocele are contiguous with each other and actually descend through the same apical transverse anterior defect. Midline defects are rare to nonexistent and the traditional distinction between cystocele and anterior enterocele is not consistent with the biodynamics of labor.

If the uterine cervix has been removed, another potential structural problem, the cervical defect (Fig. 17.1),[5,6] is created and is difficult to correct surgically and it has mechanical consequences. The pubocervical septum is shorter than the rectovaginal septum by a length equal to the diameter of the cervix. For this reason, absence of the cervix potentially increases the size of the apical anterior defect and exaggerates the effect of the disruption of the connective tissue integration in that area. Meticulous repair of the vaginal cuff at the time of hysterectomy is of primary importance in prevention of future prolapse. As discussed later in this chapter, this cervical defect creates a significant amount of


Cut edge of the peritoneum
Lig. rotundum
Lig. umbilicale mediale
Fascia umbilico-vesicalis
Fascia transversalis
Fascia vesicalis
Lig. sacro-uterinum
Fascia recti
Fascia vaginalis
Cut edge of the peritoneum
Lig. rotundum
Lig. umbilicale mediale
Fascia umbilico-vesicalis
Fascia transversalis
Fascia vesicalis
Lig. sacro-uterinum
Fascia recti
Fascia vaginalis

Suspensory axis of the uterovaginal complex

Yellow = primary
Red = anterior

Cervical defect

Peham and Amreich (modified)

Fig. 17.1 Typical pattern of endopelvic fascial damage (Reprinted with permission from Peham and Americh)

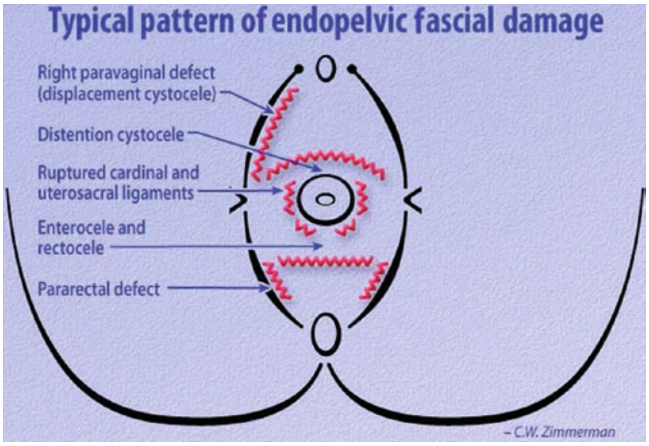

Typical pattern of endopelvic fascial damage
Right paravaginal defect (displacement cystocele)
Distention cystocele
Ruptured cardinal and uterosacral ligaments
Enterocele and rectocele
Pararectal defect
– C.W. Zimmerman

Fig. 17.2 Suspensory axis of the uterovaginal complex

difficulty for the reconstructive pelvic surgeon if normal vaginal depth, axis, and caliber are to be goals of corrective procedures.

Posterior apical transverse disruption of the rectovaginal septum occurs at the junction of that structure with the posterior pericervical ring and the uterosacral ligament.[5] Subsequent descent of the fetus under the pubic bones while following the internally concave curve of the anterior sacrum and coccyx causes distal displacement of the septum toward the perineum. The resulting defect in the continuity of the suspensory axis of the uterovaginal continuum allows both rectocele and enterocele to develop through the resulting defect.

In summary, anterior and posterior enteroceles are primarily due to obstetrical disruption of the integration of the named elements of the endopelvic fascia at their connection of the pericervical ring. The primary insult to these connections is related to the significant physical stress that is applied to this area during the progression of the fetal head as it negotiates the interspinous diameter with the cardinal movements of labor. The result of the delivery process and other subsequent physical and environmental factors is displacement of the apical avulsed edges of the pubocervical and rectovaginal septa distally. This alteration of normal anatomy leaves a weakened area both anterior and posterior that allows descent of the intra-abdominal contents through the central axis of the pelvic cavity. If the cervix has been surgically removed, these defects become contiguous, the overall size of the disruption becomes larger, and the mechanical effect of discontinuity of both arms of the suspensory axis is magnified. Surgeons who correct anterior and posterior enterocele must center their surgical efforts on restoring or compensating for endopelvic fascia connections at the level of the interspinous diameter. These connections are best appreciated by the concepts of DeLancey level I suspension and the suspensory axes of the endopelvic fascia.

Surgical Technique

Repair of Anterior Enterocele

Few areas of the body contain such an array of structurally important, functionally necessary, and surgically vulnerable structures as the interspinous diameter and the surrounding vicinity. Access to the deep pelvic structures that are important to the repair of anterior and posterior enterocele requires detailed knowledge of the anatomy of this relatively inaccessible deep pelvic region, including the avascular spaces of pelvis (see Chap. 1).

In the anterior vagina, the vesicovaginal space extends from the junction of the pubocervical septum with the urogenital diaphragm to its junction with the anterior cervix and pubourethral (bladder pillars) and cardinal ligaments. Surgical dissection of this space requires separation of the vaginal epithelium from the underlying vesicovaginal septum. This task requires access to the correct plane. Development of the vesicovaginal space begins with a midline incision through the full thickness of the vaginal epithelium taking care not to impinge on the endopelvic connective tissue or bladder both of which are deep to this dissection plane. Because of the distal displacement of the pubocervical septum, the surgeon must be particularly cautious in the apical portion of this incision especially with the initial incision. In this area, the prolapsed bladder is particularly close to the vaginal epithelium because the vesicovaginal septum does not intervene due to its avulsion and displacement. Upon incising the epithelium, one immediately encounters anterior peritoneum or visceral fascia of the bladder in the apical anterior vagina. A distinct visual difference exists between the peritoneum, bladder wall, pubocervical septum, and the vaginal epithelium that can be of valuable assistance to the surgeon (Table 17.3). Using traction and counter-traction, very fine web-like fibers can be seen that signify the presence of this plane. These fibers of Luschka are amenable to avascular dissection using a gentle push and spread technique alternating with sharp division of dense fibrous areas. Scissors that are designed for avascular space dissection are helpful such as the Yagi-Zimmerman scissors (Marina Medical, Sunrise, FL) shown in Fig. 17.3.

These instruments have a more rapidly widening blade configuration than the Mayo or Metzenbaum scissors for avascular space development. The lack of a sharp tip helps to

Table 17.3 Visual appearance of key tissues in vaginal surgery

Pubocervical septum	Collagenous, faintly white
Rectovaginal septum	Collagenous, thick, white
Bladder	Red, interlacing musculature
Rectum	Red, outer longitudinal muscle
Peritoneum	Smooth, +/– fat

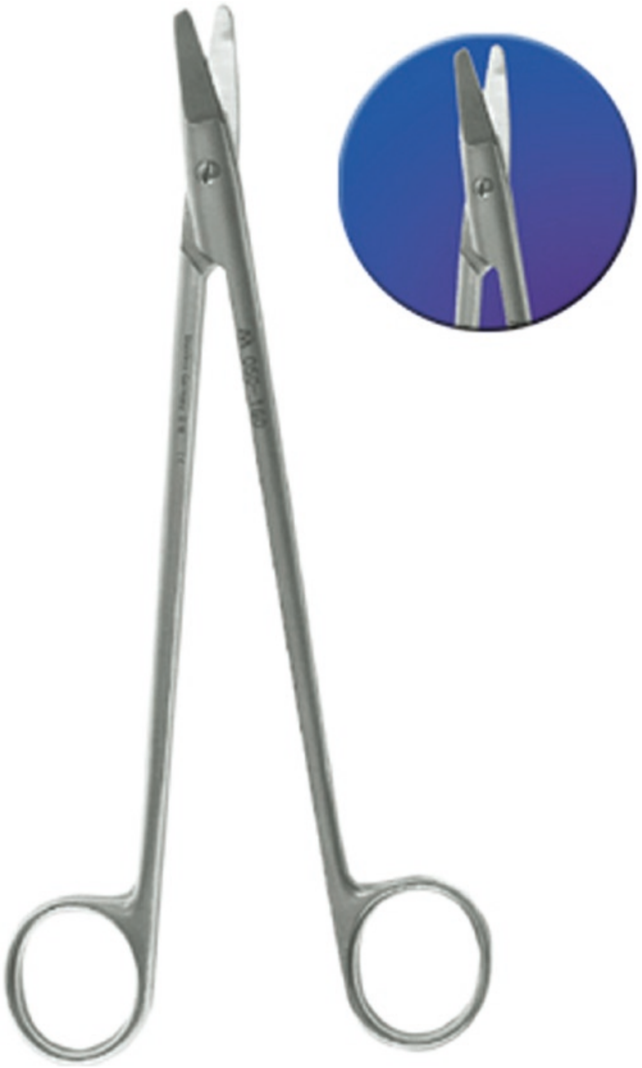


Fig. 17.3 Yagi-Zimmerman dissection scissors (Courtesy of Marina Medical Instruments, Inc., Sunrise, FL)

avoid injury to underlying structures, yet they are small enough for the limited size of the operative field.

The vesicovaginal space dissection should also extend laterally to the medial fascia of the obturator internus muscle, the location of the arcus tendineus fascia pelvis. In the anterior vagina the obturator internus fascia comprises the pelvic sidewall. Soft tissue dissection should be continued until both ischial spines can be easily palpated. If the dissection is done in the correct plane, no significant blood vessels are normally encountered if the surgery is primary. In patients who have previously been operated for pelvic organ prolapse, scarring and bleeding from the resulting neovascular reaction associated with healing can complicate the dissection and increase the amount of blood loss. If permanent mesh or a nonremodeling xenograft has been inserted in a prior surgery, the dissection process may be especially difficult or impossible to complete.

Once dissection of the vesicovaginal space is complete, the various structures involved in a site-specific repair can be identified. Identification of the pubocervical septum is a subtle skill until one has experience. It is more difficult to see than the rectovaginal septum because it does not carry as much mechanical load. For that reason, it is thinner and less substantial than its posterior counterpart. Nevertheless, it can be identified as a collagenous structure that is comprised of tropocollagen bundles. It is most easily recognized at the avulsed margins of the fascia. The visual differences between the septum, peritoneum, and visceral fascia of the bladder are most apparent in these areas because they are adjacent to each other. Irrigation with saline is helpful in both washing away blood and whitening the fascia. In the usual case, both an apical transverse (contiguous cystocele and anterior enterocele) and unilateral paravaginal paravesical defect will be present. Usually the paravaginal defect will be on the patient's right side. Recall that disruption of the septum on two adjacent sides allows for the mechanical failure of the fascial septum that allows prolapse to develop. Because the dissection extends apically to the interspinous diameter, the apical transverse edge is most apparent. After it is identified, a search can be conducted for the paravaginal defect that will usually be present on the patient's right side. Both the apical transverse edge and the paravaginal defect will be displaced from their normal anatomical location. In a high-grade prolapse, the paravaginal defect may push the edge of the septum to the contralateral side of the vagina, and the cystocele/anterior enterocele defect may retract distally to a location close to the junction of the pubocervical septum and the urogenital diaphragm.

The technique of repair should be site-specific. The basis for this type of surgery involves finding where endopelvic fascia is torn. These avulsions are commonly located at the margins of the septum, and at its junction with suspensory ligaments. After these defects are located, site-specific surgical technique is used to replace them into their normal locations and anatomical attachments. Predicated in this technique is the ability of the surgeon to find defects as outlined in the previous paragraph. Uncommonly central defects will be found. When present, central defect repair does not mechanically replace paravaginal and anterior enterocele repairs. A central repair simply restores the integrity of the septum. Likewise, an intentionally anatomically distorting midline placation is not appropriate in a site-specific repair because such a repair will only widen the lateral defect.

Paravaginal defect repair should be conducted with permanent or delayed absorbable suture. Either multifilament or monofilament suture may be chosen depending on the preference of the surgeon. Identify the ischial spine ipsilateral to the paravaginal defect. The ischial spine is the apical terminus of the arcus tendineus fascia pelvis or white line. Lateral sutures should be placed so that they encircle the white line and extend deeply into the underlying obturator internus fascia and muscle. The central side of the suture should secure the lateral edge of the pubocervical fascia defect. Several of these sutures are required to completely close the defect. At some point along the white line, the pubocervical septum will be seen to reestablish its normal connection to the white line anterior to the ischial spine marking the margin of the paravaginal defect. When these sutures are tied, the paravaginal defect is repaired and a portion of the mechanical integrity of the pubocervical septum is reestablished.

Repair of the apical anterior transverse defect that allows an anterior enterocele to develop is challenging. The most common site of failure in all pelvic organ prolapse surgery is recurrence of an anterior enterocele. Frequently, these patients have undergone a hysterectomy in the past. As noted previously in this chapter, the cervical defect prohibits a totally site-specific repair unless vaginal shortening is acceptable. Usually, restoration of normal depth, axis, and caliber are surgical priorities and therein lays the problem. The apical pubocervical septum normally connects to the pericervical ring and uterosacral ligament through the cervix. In a degree of prolapse that requires repair and especially in the absence of the cervix, the pericervical ring is not structurally intact further compounding the difficulty of creating a repair that is mechanically sound.

In a native tissue repair, the surgeon must find a secure bilateral apical suspension (DeLancey Level I) site to accomplish anterior enterocele repair. Either the sacrospinous ligaments or uterosacral ligaments can be used for this purpose. The uterosacral ligaments are the normal anatomical structure for apical suspension. If the peritoneum has been opened, the intraperitoneal portion of this structure is relatively easy to locate using the same basic technique as a McCall's culdoplasty suture. In addition, the retroperitoneal portion of the uterosacral ligament can be identified and sutured using the same landmarks and techniques that are outlined in the posterior enterocele portion of this chapter. The sacrospinous ligament terminates laterally on the ischial spine. Because dissection is already completed to that level of the deep pelvis at this point, the ischial spines can easily be identified. Placement of a permanent suture, midway between the ischial spine and the midpoint of the sacrospinous ligament can provide adequate suspension. Of course, these suspension techniques should be performed with the permanent or delayed absorbable suture of choice on both sides. If the cervix is present, the central portion of the apical transverse defect can be attached to the anterior cervix. If the cervix is absent, the hysterectomy scar or a bolster may be used to strengthen the repair as discussed below.

The apical transverse edge of the pubocervical septum will normally resist the surgeon's effort to suspend it to normal vaginal depth. As has been stated before, the absence of the cervix serves to explain this unfortunate fact. For that reason, many pelvic surgeons choose to use a bolster in this area. Several different types of materials are available for this use including synthetic thermoplastic polymers like polypropylene, autografts, and xenografts. Each of these materials has advantages and disadvantages and are discussed in other chapters of this book. The surgeon and patient should discuss and agree on any implanted material. If the decision is made to bolster a repair, it should supplement, not replace the site-specific repair. In other words, bolsters should be used to augment repairs, not as a short cut for mechanically sound surgical technique.

Repair of Posterior Enterocele

In the posterior vagina, the rectovaginal space extends from the junction of the rectovaginal septum with the perineal body near the vaginal opening to its junction with the posterior cervix and uterosacral ligaments at the level of the interspinous diameter. Surgical access to this space is gained by incising the vaginal epithelium at the level of the introitus and separating the vaginal epithelium from the underlying deep endopelvic connective tissue. The correct plane is demarcated by the presence of fibers of Luschka described in the section on anterior enterocele repair. A complete dissection of the rectovaginal space is necessary for full exposure of the rectovaginal septum, ischial spines, pararectal spaces, and retroperitoneal uterosacral ligaments.

After complete dissection of the rectovaginal space, the rectovaginal septum can be identified as a thick, whitish, collagenous septum.[5] The rectovaginal septum is connective tissue composed of collagen, elastic fibers, and a small amount of smooth muscle.[7] (Fig. 17.4) In the usual case, the septum is contiguous with the perineum and extends part of the way

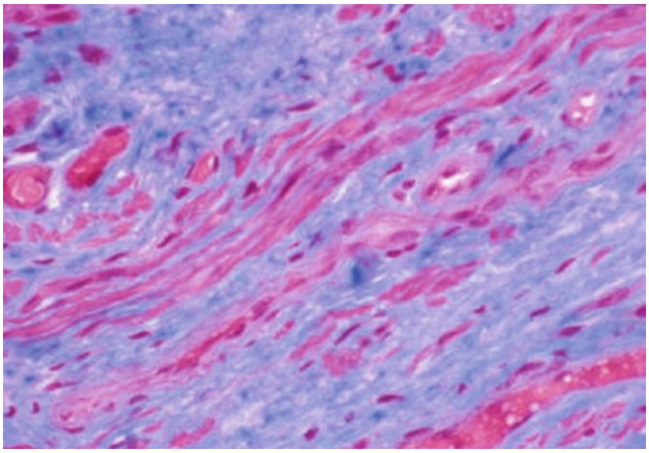


Fig. 17.4 Histology of the rectovaginal septum (Masson Trichrome Stain 40×, elastic fibers: red, collagen: fibers blue)

toward the interspinous diameter. The septum is easiest to identify at its apical transverse margin. The edge of this septum will be more apparent than the low load-bearing vesicovaginal septum. Irrigate the surgical site with saline. A clearly visible transition will occur between the rectovaginal septum and the smooth surface of the peritoneum and retroperitoneal fat. The surgeon may also be able to see the outer longitudinal smooth muscle fibers of the rectal wall in the distal part of the defect. Posterior enterocele and rectocele protrude through the same fascial defect. Grasp the apical edge of the rectovaginal septum with Allis clamps and the entire septum will become easier to identify. The rectovaginal septum should be freed of the vaginal epithelium and any secondary adhesions all the way laterally to the superior fascia of the pelvic diaphragm over the levator ani muscles. In the posterior vagina, the levator ani muscles mark the pelvic sidewall. The surgical goal for the rectovaginal septum is elevation to the interspinous diameter and mechanically sound apical suspension.

Dissection should continue apically until both ischial spines can be easily palpated. These bony structures will allow identification of the pararectal spaces located medial and deep, i.e., posterior to the spines on each side. The structurally intact retroperitoneal portion of the uterosacral ligaments can be found at the top or cranial portion of the pararectal spaces. A thoracic length long Allis clamp may be used to grasp the uterosacral ligament in this location. Lighting and irrigation are essential for this task as with the Versalight™ instrument (Lumitex MD, Strongsville, OH). Prior to the development of posterior prolapse, the uterosacral ligament has been avulsed from the pericervical ring and rectovaginal septum. In the presence of a rectoenterocele, the uterosacral ligaments are not attached distally, and, for that reason, cannot be palpated prior to grasping them. Because of concern for the rectum, and because the uterosacral ligaments can be palpated when grasped and traction is applied, some surgeons prefer to perform a rectal exam during this exercise. With a finger in the rectum, the ligament can be felt, when grasped, as it courses toward the sacrum. To the naked eye, the ligament does have the appearance of collagenous tissue; however, it is not always easy to see during the learning curve required to identify these structures. On average, the ureter is approximately 3 cm cranial to this portion of the uterosacral ligament.[8-10] Additionally, the portion of the uterosacral ligament that can be identified within the pararectal space and ureter are separated by the dense connective tissue of the paracolpium, and, for that reason, it is relatively safe from injury. Even with the relatively great distance between the ureter and the uterosacral ligament that can be identified within the pararectal space, cystoscopy to ensure ureteral patency should be performed after this type of procedure (Fig. 17.5). Care should be taken to remain medial to the ischial spines because the internal pudendal artery and veins, pudendal nerve, and the lumbosacral plexus of nerves exit the pelvis through the greater sciatic notch. A double pass helical permanent suture should be placed in each ligament. To pass this suture, a needle holder or mechanical suturing device may be used.

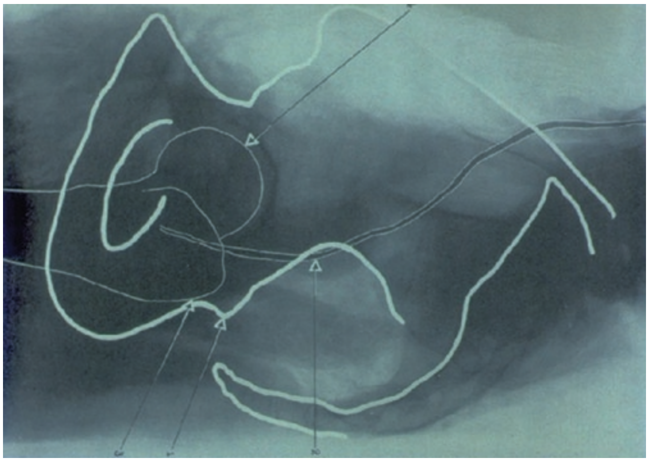


Fig. 17.5 X-ray of a cadaver pelvis with contrast material in the ureter and key bony structures outlined. (Reprinted from Uhlenhuth[10]. With permission from Lippincott Williams & Wilkins)

The uterosacral ligaments function as the primary apical suspensory elements for the entire uterovaginal complex and bear the load of both the anterior and posterior arms of the suspensory axis in normal female pelvic anatomy. This fact makes them the ideal apical suspensory structure. They can be accessed bilaterally to distribute the mechanical load. Using the uterosacral ligaments in this way creates a vaginal uterosacral colpopexy that creates the same effect on suspension as the abdominal sacral colpopexy without the laparotomy or laparoscopy.

The sacrospinous ligaments are an acceptable alternative for apical suspension. They are easily located as palpable fibromuscular structures that have their origin on the lateral sacrum and insertion on the ischial spine. The technique of sacrospinous ligament fixation has been well-described and is widely practiced. Mechanical suturing devices, like the Capio™ (Boston Scientific, Natick, MA), are popular for this technique because of the limited surgical exposure deep in the pelvis. Care should be taken not to injure the rectum. The advantages of sacrospinous ligament fixation include strength of the ligament, potential for bilateral suspension, and ease of identification of the target structure. The disadvantages include potential for shortening the vagina and posterior deviation of the vagina that places some stress on the area of the cervical defect in the anterior vagina. The primary advantage of the uterosacral ligaments for apical suspension is the fact that it is the normal anatomical attachment point on each side of the apical vagina.

If the uterosacral ligament or sacrospinous ligaments are not used for apical suspension during prolapse surgery performed vaginally, the surgery has a high risk of failure. Synthetic mesh, xenografts, or autografts used in abdominal sacral colpopexy are examples of acceptable substitutes for apical suspension when the operation is performed abdominally.

To complete restoration of the suspensory axis, the apical transverse edge of the rectovaginal septum should be attached to the uterosacral or sacrospinous ligaments bilaterally. This step restores the continuum of connective tissue between the perineum and the presacral periosteum. This uterosacral colpopexy also simultaneously corrects rectocele, enterocele, and perineal descent, and often has a considerable positive effect on anal prolapse. At this point, only two sutures have been described in this surgery. Other steps may be taken to further strengthen the repair. One option is to place a bolster of polypropylene within the interspinous diameter using either a trocar insertion technique or simply suturing a bolster into place with the uterosacral ligament colpopexy sutures. This step places a sling within the interspinous diameter, the plane of maximum damage in prolapse. A scaffolding that substitutes for the destroyed pericervical ring and is available for placement of sutures that help distribute the load of suspension is created. As seen in Fig. 17.6, three equally spaced sutures may be placed centrally in the posterior reconstruction to complete rectocele and enterocele repair. Anteriorly, the central apical sutures can also be attached to this scaffolding if desired (Fig. 17.6). When all of these steps are completed, the endopelvic connective tissues are reintegrated in a site-specific anatomically restorative way deep within the pelvis.

Many surgeons prefer the use of bolsters in pelvic reconstructive surgery. These strengthening materials are available in various materials including autografts, polypropylene, cross-linked xenografts, and noncross-linked xenografts that are biochemically intact and have the ability to remodel over time. The United States Food and Drug administration has issued a warning that alerts surgeons and patients to potential risks of these materials.[11] Bolstering of ventral hernia repairs is known to decrease the likelihood of recurrence. No definitive data is available to demonstrate that the same outcome improvement is present in vaginal prolapse surgery. Certainly good studies are needed. Until that time, surgeons and patients should proceed with caution, common sense, and informed consent. Certainly, from a surgeon's perspective, it is unlikely that any implanted material can compensate for a technique that is not mechanically sound (Fig. 17.6).

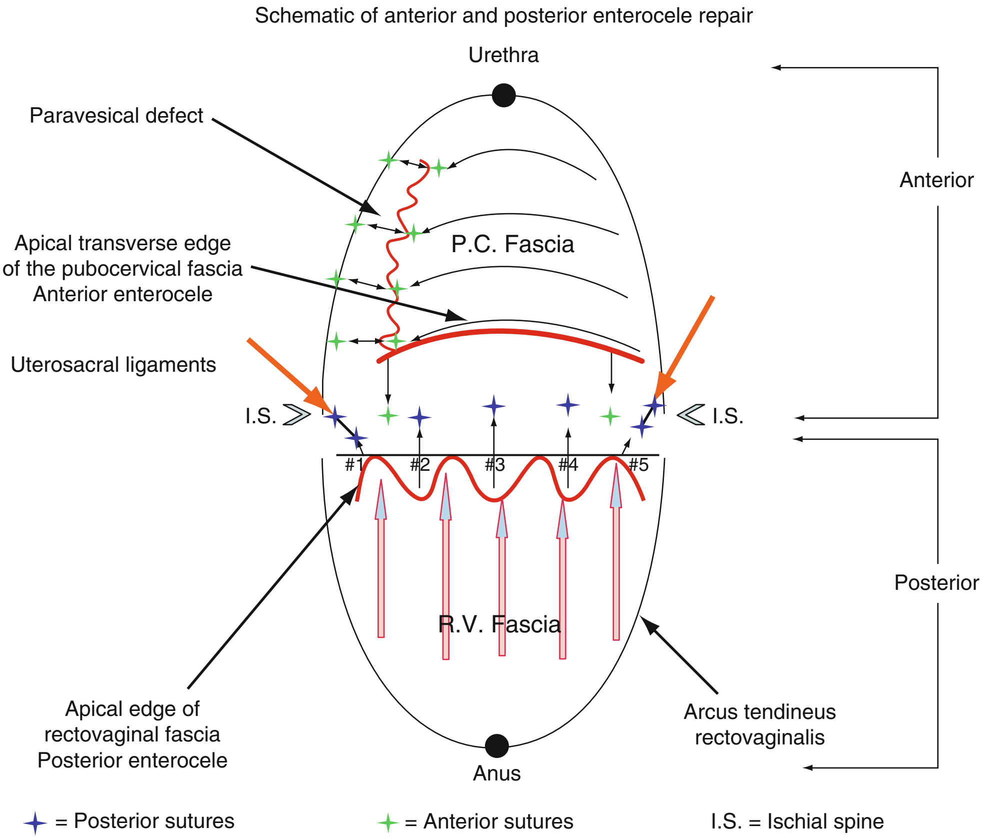

Schematic of anterior and posterior enterocele repair
Urethra
Paravesical defect
Anterior
P.C. Fascia
Apical transverse edge of the pubocervical fascia Anterior enterocele
Uterosacral ligaments
I.S.
I.S.
#1
#2
#3
#4
#5
Posterior
R.V. Fascia
Apical edge of rectovaginal fascia Posterior enterocele
Arcus tendineus rectovaginalis
Anus
✦ = Posterior sutures
✦ = Anterior sutures
I.S. = Ischial spine

Fig. 17.6 Schematic of anterior and posterior enterocele repair including restoration of the suspensory axis

Conclusion

In many ways, the techniques required for correction of anterior and posterior enterocele are central to prolapse repair in general. Connective tissue damage from childbirth is centered within the interspinous diameter and an effective repair should also be centered in that surgically challenging location. Repairs that are anatomically distorting or that excessively rely on bolsters and plication rather than anatomically restorative and biomechanically sound techniques are likely to have worse anatomical and functional outcomes. Another obvious concept is that it is impossible to correct a problem that is centered deep in the central pelvis by plicating tissues in the distal vagina. Patients deserve to have well-designed surgeries that maximally restore form and function to the vaginal vault.

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Part V

Posterior Compartment Repair

Treatment of Posterior Vaginal Wall Defects

18

Carl W. Zimmerman and Karen P. Gold

Introduction

As life expectancies increase and the population ages, an increasingly greater number of women will require surgery for pelvic organ prolapse. Over a decade ago, Olsen et al. reported an 11.1% lifetime risk of undergoing surgery for pelvic organ prolapse or urinary incontinence by age 80. Of these surgeries, 44.5% included posterior compartment repair and 29.2% were reoperation.[1] Hendrix et al. evaluated the prevalence of pelvic organ prolapse in women enrolled in the Women's Health Initiative; rectocele was found in 18.6% of the 16,616 women with a uterus and 18.3% of the 10,727 women who had a hysterectomy.[2] More recently, Nygaard et al. demonstrated a weighted prevalence of at least one pelvic floor disorder in 23.7% of 1,961 women in a cross-sectional analysis, of which 15.7% experienced urinary incontinence, 9.0% fecal incontinence, and 2.9% pelvic organ prolapse. As noted in previous studies, the proportion of women who reported symptoms of at least one pelvic floor disorder increased with age.[3]

Defects of the posterior vagina include rectocele, enterocele, perineal descent, and perineal attenuation. A rectocele is defined as a bulge, prolapse, or herniation of the anterior rectal wall through the posterior vagina.[4] An enterocele is a posterior vaginal hernia containing small intestine and the lining of the peritoneal cavity that protrudes through the posterior cul-de-sac.[5] Perineal descent refers to increased downward mobility of the perineal body, which usually lies within 2 cm of an imaginary line between the ischial tuberosities,[6] whereas perineal attenuation is a disruption of the perineal body and is commonly obstetric, iatrogenic, or secondary to incomplete or faulty repair.

Symptoms associated with posterior vaginal defects include those associated with the process of herniation, defecatory complaints, and sexual dysfunction. The process of herniation of the posterior vagina can lead to symptoms such as pelvic heaviness or vague abdominal discomfort, usually without pain. A woman may complain of feeling or seeing something bulging or falling out of the vaginal area if the defect extends beyond the hymen; symptoms are much more likely when the bulge extends beyond the hymenal ring.

Bowel complaints include defecatory urgency or discomfort, feeling of incomplete evacuation, also known as obstructed defecation syndrome, rectal protrusion during or after defecation such as rectal prolapse or intussusception, and incontinence of flatus or stool. Some women may even report using digital manipulation or splinting of the vagina, perineum, or anus to complete defecation; in fact, this is the defecatory symptom reported most consistently.[7–10] A very large posterior vaginal prolapse may cause a mechanical obstruction leading to urinary retention.[11]

Sexual dysfunction may affect 40–60% of normal couples, but an increased risk has been linked to pelvic organ prolapse.[12,13] Recently, studies have revealed that vaginal anatomy (caliber, length, and atrophy) does not correlate well with sexual function.[14] However, an increasing grade of prolapse does predict interference with sexual activity.[15]

Etiology

Multiple factors have been associated with pelvic organ prolapse; those with the strongest correlation include childbirth, obesity, and aging. Other contributory factors may include ethnicity, congenital or acquired connective tissue disorders, neurologic injury to the pelvic floor, chronic constipation, diabetes, and chronic conditions which increase intra-abdominal pressure.[2,3,16–20]

Two studies reported risk factors for pelvic organ prolapse in women enrolled in the WHI study. Bradley et al. demonstrated that obesity and multiparty were significant risk factors for a subgroup of 259 postmenopausal women for progression of vaginal descent over a 4-year period.[17] Hendrix et al. evaluated 27,342 women, and found all sites of pelvic organ prolapse to be higher among older women, a BMI of

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25–30 kg/m^2 to be associated with an increase in rectocele of 38%, and a BMI of >30 kg/m^2 to be associated with an increase in rectocele of 75%.[2]

In a recent study for the Pelvic Floor Disorders Network, Nygaard et al. evaluated the effect of age on pelvic floor disorders; the proportion of women with at least one pelvic floor disorder increased incrementally with age.[3]

The substantial forces associated with childbirth have been strongly linked to pelvic organ prolapse.[13,16,17,21] The Oxford Family Planning Association Study declared parity as the strongest risk factor for pelvic organ prolapse with an adjusted risk ratio of 10.85.[16] If the effects of the mechanical forces of childbirth are analyzed, one can better understand the cause and usual location of damage to the deep endo pelvic connective tissues.[21–23] As the fetal presenting part progresses through the seven cardinal movements of labor, significant pressure and strain is placed on the connective tissues of the pelvis, particularly in the area of the interspinous diameter. The interspinous diameter is the narrowest pelvimetric measurement in the human pelvis, and also where the junction of the apical rectovaginal septum joins the pericervical ring and uterosacral ligaments.

After undergoing engagement, descent of the fetal vertex is followed most commonly by internal rotation from left or right occipitoanterior to occipitoanterior for clearance of the interspinous diameter at the level of the ischial spines. Flexion also occurs to allow the fetal head to pass under the pubic arch anteriorly. This combination of movements puts tremendous strain on the junction of the rectovaginal septum with the pericervical ring and uterosacral ligaments. As the fetal head continues descent, extension allows passage over the internal concavity of the sacrum and coccyx as well as further clearance under the pubic arch. The net result of these fetal maneuvers is displacement of endopelvic connective elements away from the interspinous diameter.

Frequently, separation of the rectovaginal septum from the uterosacral ligaments and pericervical ring within the interspinous space occurs. This disruption of the upper posterior rectovaginal septum creates the potential for future development of rectocele, enterocele, and perineal descent, whereas the disruption of the lower vagina and perineum results in a lower rectocele and perineal deficiency. These latter two lesions are due to perineal trauma at the time of delivery, not the passage of the fetus through the interspinous diameter.[21]

Anatomy

Pelvic anatomy is covered in detail in Chap. 1; therefore, only the posterior vaginal compartment anatomy necessary to understand the surgical repairs described here are reviewed.

The fibroelastic connective tissue layer between the vagina and rectum has been named the rectovaginal septum and has been referred to, by some, as the rectovaginal fascia. The definition of fascia is a sheet or band of fibrous connective tissue enveloping, separating, or binding together muscles, organs, and other soft structures of the body.[24]

Historically, considerable debate has occurred regarding the exact nature and function of the rectovaginal septum and its proper place, as a native tissue element in the repair of posterior vaginal defects. Richardson addressed this idea in 1993, stating "regardless of its embryologic origin and regardless of the term chosen for it, almost all gross anatomists who have studied the pelvic connective tissue have been able to demonstrate a layer of strong tissue immediately under the posterior vaginal mucosa that separates the dorsal rectal compartment from the ventral urogenital compartment in both men and women."[25]

In 1839, Denonvilliers, a French anatomist, described a perivesical two-layered fascia in the male called the rectovesical fascia, since noted as "Denonvilliers' fascia." Subsequently, authors began calling what they believed to be an analogous structure in the space between the vagina and rectum "Denonvilliers' fascia" in the female.[25] In 1954, Ricci and Thom cited evidence refuting the existence of "fascial tissue" in the integrity of the vaginal walls. This was based entirely on the study of hematoxylin- and eosin-stained histologic preparations and involved no correlation with gross anatomic dissections.[26] In 1957, Uhlenhuth and Nolley cited evidence from gross anatomic dissection and disagreed with Ricci and Thom.[27] Nichols and Milley, in 1968, combined both gross anatomy and related histologic specimens, and demonstrated a rectovaginal septum that could be identified as a distinct and relatively strong connective tissue layer between the vagina and the rectal walls. The tissues of this septum are always adherent to the posterior aspect of the vaginal connective tissue, but may easily be separated from it by blunt dissection. The demonstrable adherence to the "vaginal wall" would seem to explain, at least partially, why the existence of a septum has, at times, been denied by some authors and surgeons[28] (Fig. 18.1).

More recently, Ichiro Nagata et al. evaluated the entire vagina and its adjacent anterior wall of the rectum (i.e., rectum–vaginal interface tissues) from 20 postmortem female cadavers. The rectovaginal septum was defined as an elastic fiber-rich plate along the posterior vaginal wall. It lines the posterior surface of the vein-rich zone of the vaginal wall and extended apically to the area between the structures of the paracolpium. The septum was more evident in the lower half of the interface than in the upper half; however, they did not clearly distinguish between parous and nonparous subjects. Parity may account for the apical attenuation of the rectovaginal septum in some of their preparations. The recto vaginal septum was often thin and interrupted. Since the

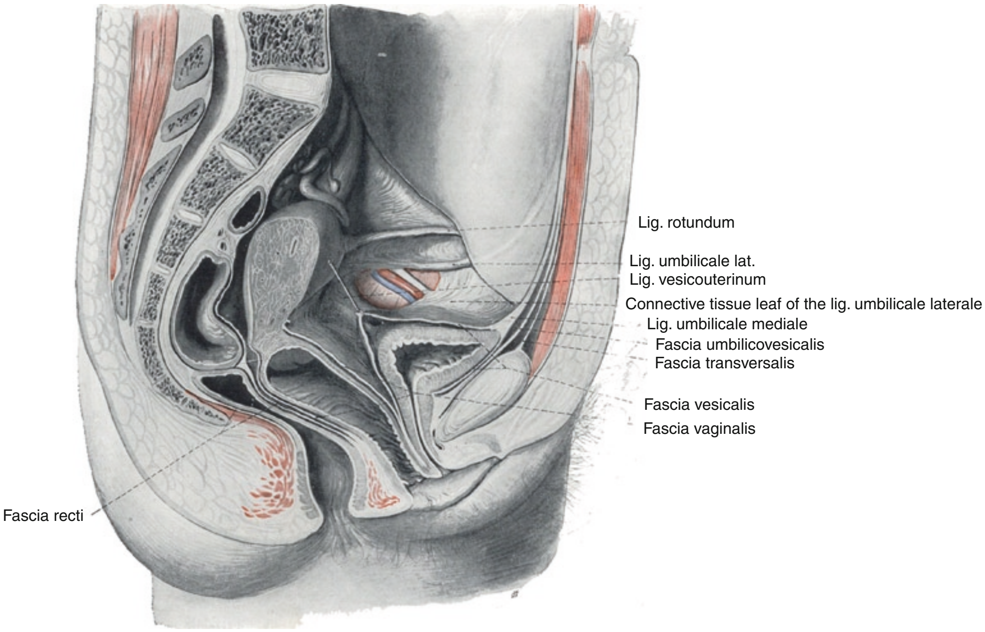

Fig. 18.1 The rectovaginal septum (Reprinted from Peham and Amreich,[29] Figure 139. With permission)

rectovaginal septum was not so clearly demonstrated in the upper vagina histologically, augmentation using some implant was considered by them to be necessary for the enterocele and high rectocele. Surgical procedures for low rectocele repair should be individualized, since the thickness and tightness of the rectovaginal septum in the lower vagina vary with the patient[30] (Fig. 18.2).

Delancey's well-known description of the biomechanical levels of posterior vaginal anatomy includes three levels of support. The apical vagina is dependent on suspension to the presacral periosteum via the uterosacral ligaments. The mid-vagina is attached laterally to the superior fascia of the pelvic diaphragm at the arcus tendineus fasciae pelvis and the arcus tendineus fasciae rectovaginalis. The distal vagina fuses with the proximal perineal body posteriorly and the urogenital diaphragm anteriorly.[31]

The primary suspensory axis of the uterovaginal complex courses along the posterior vagina from the perineum, through the rectovaginal septum, past the posterior pericervical ring, and along the uterosacral ligaments to its insertion into the presacral periosteum overlying sacral vertebrae 2, 3, and 4.[23,26–28] Vaginal support arises from interactions between the pelvic musculature and connective tissue.[31] Muscular support in the posterior compartment is provided by the pelvic diaphragm, a paired group of muscles including the puborectalis, pubococcygeus, iliococcygeus (levator ani), and coccygeus. The deep endopelvic connective tissue is made up of the uterosacral ligaments, cardinal ligaments, pubocervical ligaments, pubocervical septum or fascia, rectovaginal septum or fascia, and the pericervical ring.[32] Loss of muscular support via damage or denervation places more of the normal intra-abdominal pressure directly on the structures of the endopelvic connective tissue, which results in attenuation or tearing of the connective tissue over time (Table 18.1).

Most patients with symptomatic pelvic organ prolapse will have damage to the anterior, posterior, and superior segments of the vagina.[33] All of the vaginal segments are interconnected, and their support is interdependent.[26–28] With the exception of perineal deficiency, signs and symptoms of posterior vaginal prolapse are a result of the apical disruption of the rectovaginal septum. This disruption initially leads to the characteristic bulging in the posterior vaginal wall, and is normally associated with development of a rectocele, enterocele, and perineal descent (Fig. 18.3).

In order to choose the best treatment and hope for the best outcomes, the entire nature of the defect must be fully understood. Posterior vaginal repairs may fail because correction of defects in the upper vagina and rectovaginal septum to the cardinal/uterosacral ligaments and pericervical ring are not repaired adequately. Any successful pelvic reconstructive surgery must be designed to account for all three levels of vaginal support and attachment and restoration of the suspensory axis of the vagina.[23]

Treatment

Treatment for posterior vaginal wall prolapse should be guided not only by anatomical findings, but, more importantly, by patient symptoms. Diagnosis of a posterior vaginal defect requires a thorough evaluation, including a complete history and a physical examination. In patients with significant defecatory symptoms, anal manometry and/or defecography

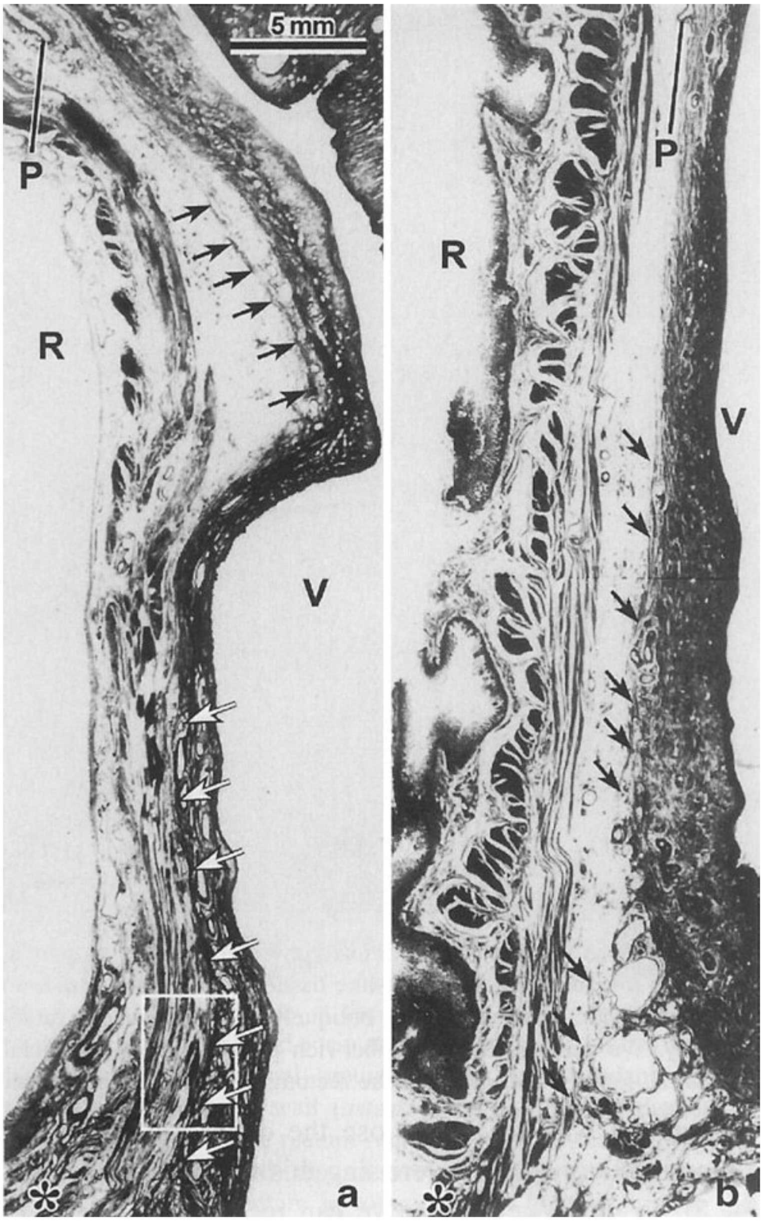
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Fig. 18.2 Sagittal sections of the rectum–vagina interface. (**a**) (73-year-old, para – two both vaginal deliveries) contains a vein-rich zone and a thick elastic fiber-rich plate (*white arrows*). However, the plate is thin and interrupted in the upper part of the interface (*black arrows*). (**b**) (88-year-old, para – unknown) displays a wide areolar tissue at the rectum–vagina interface and a thin and interrupted elastic fiber-rich plave (*black arrows*). A bulky venous plexus is evident in (**b**). Hematoxylin and eocin staging. *P* (or *asterisk*) indicates the peritoneal reflection at Douglas' pouch (or the upper edge of the internal anal sphincter). Higher magnification views of a squared area in a. *R* lumen of the rectum, *V* lumen of the vagina (Reprinted from Nagata et al.[30] Figure 1. With permission. Copyright © 2007 American Medical Association. All rights reserved)

Table 18.1 Posterior suspensory axis

Perineum
Rectovaginal septum
Pericervical ring
Uterosacral ligaments
Presacral periosteum

should be considered; if an anal sphincter defect is suspected in patients with fecal incontinence, an endoanal ultrasound is recommended. Often findings on physical examination do not correspond with the patient's symptoms, and, more importantly, her quality of life as reflected by her level of annoyance or discomfort. Many women with defects of the posterior vaginal axis do not experience symptoms and do not need

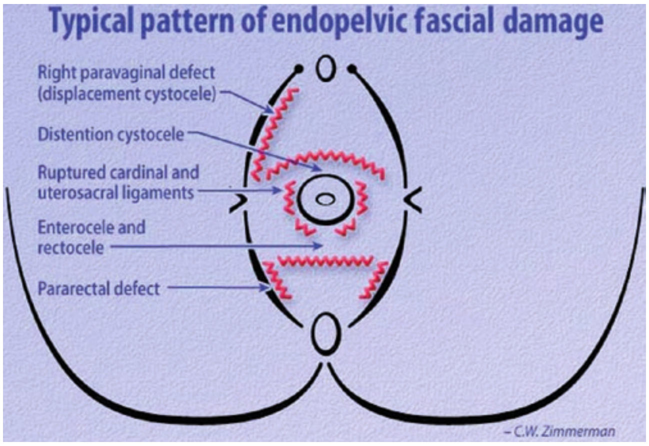
Typical pattern of endopelvic fascial damage
Right paravaginal defect (displacement cystocele)
Distention cystocele
Ruptured cardinal and uterosacral ligaments
Enterocele and rectocele
Pararectal defect
– C.W. Zimmerman

Fig. 18.3 Zimmerman POP map

treatment. The only symptom specific to prolapse is an awareness of a vaginal bulge or protrusion; resolution with treatment of any other symptoms reported by the patient cannot be assumed.[7–10]

Nonsurgical Treatment

Nonsurgical treatments of posterior vaginal defects include observation, pessaries, pelvic floor physical therapy, and treatment of symptoms. Pessaries should be offered to all women as first-line treatment and an alternative to surgery.[21] Physical therapy and biofeedback have been used with varying degrees of success in patients with posterior vaginal prolapse. Treatment of symptoms can include diet modification such as increasing water and fiber intake, physical exercise, stool bulking agents, and laxatives. Management of defecation with splinting is a helpful technique that should be taught to patients. Splinting consists simply of asking the patient to manually support the perineum during defecation and is distinct from digital evacuation. Even if surgery is performed, splinting may help avoid excessive stress on the posterior vaginal structures during defecation, especially if that event is assisted by Valsalva's maneuver. Splinting can assist in retention of pessaries during defecation and can help avoid strain on surgical repairs during the healing process.

Surgical Treatment

Women with symptomatic posterior vaginal prolapse who choose not to or are unable to use nonsurgical options are candidates for surgery. Surgical procedures include both reconstructive and obliterative techniques. Surgical procedures have included transvaginal, transanal, abdominal, and laparoscopic approaches.

The operative goal of posterior pelvic prolapse should include repair of central, lateral, proximal (apical), and distal defects. Central defects should be corrected by repairing all defects in the rectovaginal septum. Lateral defects require reattachment of the rectovaginal septum to the superior fascia of the levator ani muscles via the arcus tendineus fascia pelvis and arcus tendineus rectovaginalis. Proximal or apical defects are repaired by both reattachment of the rectovaginal septum to the uterosacral ligaments or the sacrospinous ligaments laterally and the pericervical fascial ring centrally. Distal defects require the establishment of fusion of the rectovaginal septum with an appropriately reconstructed perineal body. Repair of an isolated vaginal segment in the presence of unrecognized or incipient damage to another segment is clinically inappropriate. Care must be taken to fully assess all aspects of pelvic support when planning a procedure.[23]

The original procedures described for posterior vaginal wall defects narrowed the caliber of the vagina in an intentionally anatomically distorting effort to correct and prevent further development of pelvic organ prolapse.[32] This type of procedure includes the traditional posterior colporrhaphy described by Francis and Jeffcoate. This method of posterior repair with levator ani plication has been abandoned by reconstructive vaginal surgeons due to its propensity to cause dyspareunia.[34–36] To minimize the risk of dyspareunia, site-specific repair and posterior colporrhaphy (midline fascial plication) without levator ani plication have been utilized with anatomic success rates of 77–100%, although functional outcomes are not as consistent and postoperative dyspareunia rates of 8–26% have been reported.[25,36–41]

Maher et al. prospectively evaluated 38 women with symptomatic rectocele who underwent posterior colporrhaphy (midline fascial plication without levator ani plication), objective success rates were 87% at 12 months and 79% at 24 months. This study also found an association between anatomic defect correction and improved functional outcome; 87% of the women had resolution of obstructed defecation and significant improvements were also seen in straining to defecate, hard stools, and dyspareunia.[37] Ambramov et al. compared 124 women with site-specific repair and 183 women with posterior colporrhaphy without levator plication in a retrospective chart review with at least 1 year of follow-up. The site-specific repair group had a significantly higher rectocele recurrence rate compared to the posterior colporrhaphy group; however, the patients were not randomly assigned, and selection bias may have influenced the outcomes.[42] Cundiff et al. reported on a prospective case series of 69 women who underwent discrete defect repair in the rectovaginal fascia; 82% had an anatomic success rate at 12 months, and significant improvements were seen in sexual function and bowel symptoms.[43]

The Cochrane review by Maher et al. confirms the lack of randomized trials that include surgical operations for posterior vaginal wall prolapse.[44] They identified and reviewed only four randomized or quasirandomized trials. Two trials compared vaginal and transanal approaches for the management of rectocele.[45] The results for posterior vaginal wall repair were better than for transanal repair in terms of subjective and objective failure rates (RR 0.24, 95% CI 0.09–0.64), although there was a higher blood loss and postoperative narcotic use. Sand et al. (2001) compared posterior repair with and without mesh reinforcement. Rectocele recurrence appeared equally common with and without polyglactin (Vicryl) mesh augmentation (7/67 vs 6/65).[46] In 2006, Paraiso et al. compared posterior colporrhaphy ($n = 28$), site-specific repair ($n = 27$) and site-specific repair augmented with porcine small intestine submucosa graft inlay (Fortagen, Organogenesis) ($n = 26$) for repairing rectocele. There was no significant difference between the three groups in subjective functional failures (15% overall) or symptomatic outcomes in this comparison; however, the group receiving graft augmentation had a significantly greater anatomic failure rate (46%) than those who received site-specific repair alone (22%) or posterior colporrhaphy (14%).[41]

Augmentation of the rectovaginal space with both biologic grafts and synthetic meshes has been attempted with mixed outcomes. Biologic grafts include allografts (human donor), autografts (self donor), and xenografts (animal donor). The most commonly used synthetic mesh is poly propylene. Anatomical and functional outcomes may be more dependent on proper surgical technique than specific types of augmentation.

The use of synthetic mesh to augment repair of the posterior compartment has not shown the anticipated success, and even more concerning are the complications exhibited.[46–50] In 2005 at the World Health Organization's third International Consultation on Incontinence, it was recommended that mesh placed transvaginally be done so only in well-designed clinical trials and not in general practice until more data is available. On October 20, 2008, the FDA released an alert to physicians concerning the potential risks of transvaginal surgical mesh used to treat pelvic organ prolapse and stress urinary incontinence.[51] The most frequent complications included erosion through vaginal epithelium, infection, pain, urinary problems, and recurrence of prolapse and/or incontinence. There were also reports of bowel, bladder, and blood vessel perforation during insertion. In some cases, vaginal scarring and mesh erosion led to a significant decrease in patient quality of life due to discomfort and pain, including dyspareunia. Recommendations were given to help reduce the risk of possible complications. The Society of Gynecologic Surgeons has formed a systematic review group to develop evidence-based guidelines on biologic and synthetic graft use compared with native tissue repair in vaginal prolapse repair. Based on the overall low quality of evidence, only weak recommendations could be provided on the basis of available data. They suggested that native tissue repair remains appropriate when compared with biologic graft, absorbable synthetic graft, and nonabsorbable synthetic graft in the posterior compartment.[52]

When considering repair of the posterior compartment, one must not overlook the importance of the suspensory axis. Delancey's Level I concept is central to reestablishing the integrity of the suspensory axis. These operations must be centered within the interspinous diameter, not in the middle or distal vagina, as plication procedures have traditionally been done. The abdominal sacral colpopexy has been called the gold standard for suspension of the vaginal apex and has been demonstrated to have a lower rate of recurrent vault prolapse than vaginal sacrospinous colpopexy and less dyspareunia. However, the vaginal approach takes less time and allows for a more rapid recovery without the possible complications associated with abdominal surgery and intra-abdominal implants.[53,54]

We propose a comprehensive repair of the posterior vaginal defect, including central, lateral, proximal (apical), and distal deficiencies. Considering the available literature on repair of the posterior compartment, both anatomical and functional outcomes have to be considered. The three levels of vaginal support proposed by DeLancey must be addressed, and the suspensory axis of the vagina must be restored. Surgical progression should proceed from apical to distal. Posterior vaginal reconstruction should begin with DeLancey Level I suspension, then progress to DeLancey Level II lateral attachment if required by the patient's defect. After these two levels of anatomy have been corrected, DeLancey Level III distal fusion should be addressed, if necessary.

Posterior repair must include full-length and full-width rectovaginal septum reconstruction, with site-specific repair of central defects, central enterocele and rectocele closure, anatomic perineal reconstruction, and bilateral vaginal uterosacral or sacrospinous colpopexy. The vaginal uterosacral colpopexy reestablishes the normal anatomic connection between the rectovaginal septum and both the uterosacral ligaments and the pericervical ring. In his early description in 1997 of uterosacral ligament fixation, Jenkins evaluated 50 women with vaginal vault prolapse in whom he was able to successfully identify and utilize the uterosacral ligaments to suspend the apical vagina without subsequent failure or significant complications as observed over a 4-year follow–up period.[55] The use of uterosacral vault suspension has been shown to be anatomic and durable, as well as maintaining or improving the urinary, bowel, and sexual function of the vagina.[56–58] Alternative apical attachment sites, such as the sacrospinous ligament or iliococcygeus fascia, may be used if the uterosacral ligament is unavailable.[59–63]

Operative repair begins with a posterior midline incision of the vaginal epithelium and is extended superiorly as far as necessary to allow adequate exposure of the interspinous diameter and access to the pararectal space. Complete dissection of the rectovaginal space from the perineum to the level of the ischial spines is carried out. Sharp dissection is begun at the medial edge of the vaginal epithelium to carefully enter the mostly avascular plane separating the epithelium from the underlying rectovaginal septum. Once in the proper plane, blunt dissection should proceed smoothly and quickly to the boundaries of the rectovaginal space, including the superficial fascia of the pelvic diaphragm or pelvic sidewall. Sharp dissection may be required in areas of connective tissue bands or adhesions from previous surgical attempts at repair. At the 3 and 9 o'clock positions, the vaginal arterial and venous vessels may be encountered. Apically, the dissection must extend to the level of the ischial spines. It is here at the level of the interspinous diameter that the uterosacral ligaments necessary for reconstruction of the suspensory axis of the vagina can be identified within the pararectal spaces.

Following complete dissection of the rectovaginal space, the elastic fiber-rich plate described by Nagata et al. as the rectovaginal septum can be identified.[30] The apical edges of the rectovaginal septum can be grasped with Allis clamps in preparation for suspension to the uterosacral ligaments. Immediately apical to the edge of the rectovaginal septum, a rectocele may be identified by the longitudinal fibers of the muscular wall of the rectum. An enterocele in the most apical rectovaginal space can be seen as very thin and smooth tissue with the characteristic yellow color of retroperitoneal fat. In advanced cases of prolapse, the rectovaginal septum that is torn transversely from the uterosacral ligaments and pericervical ring may be retracted all the way to the level of the perineum. Another common pattern of fascial damage is an intact connection of the septum to the uterosacral ligaments on one side and a complete separation with a full length pararectal defect on the side with the uterosacral disruption. This pattern of rectovaginal septum damage often results in retraction of the septum to the intact side of the septum. Following complete dissection of the rectovaginal septum, the uterosacral ligaments must be identified; the key landmark for this operative step is the ischial spines (Figs. 18.4 and 18.5).

Buller et al. evaluated female cadavers to identify the optimal site in the uterosacral ligament for suspension of the vaginal vault with regard to adjacent anatomy and suspension strength. The intermediate portion of the uterosacral ligament was found to be 2.3 ± 0.9 cm from the ureter, and the distance from the ischial spine to the ureter was 4.9 ± 2.0. The ischial spine was found consistently beneath the midportion of the uterosacral ligament. The uterosacral ligament supported 17 kg of weight before failure when strength testing was performed.[64] If a thorough dissection has been completed bilaterally to the ischial spines, a Heaney or Breisky retractor can be placed adjacent to the ischial spine and downward traction can be applied to expose the pararectal space. Visualization can be significantly improved with the aid of a lighted suction irrigator such as the VersaLight (Lumitex, Strongsville, OH). Once the spine is palpated and the pararectal space is visualized, a long Allis clamp is used to grasp the uterosacral ligament in a side-to-side fashion just medial and cephalad to the ischial spine. Access to the uterosacral ligament is via the pararectal space in the retroperitoneum. This portion of the uterosacral ligament is minimally impacted by the forces of childbirth as noted in the etiology section of this chapter. Once grasped, this ligament is often strong enough to actually move the patient on the table. If uterosacral ligaments are not easily identified, an examining finger can be placed in the rectum and palpation of the uterosacral ligament beside the rectum can be accomplished when the ligament is placed on tension. Once appropriately identified and grasped with a long Allis clamp, a double-pass permanent suture is placed into each of the uterosacral ligaments. The Capio device (Boston Scientific, Natick, MA) or a similar device is helpful with the placement of these sutures because access is limited in this deep space. The authors prefer braided polyester (Ethibond) for this step; however, alternatives such as silk, nylon, or monofilament polypropylene are also acceptable. Some surgeons prefer delayed absorbable sutures, although the ventral hernia literature clearly endorses the use of permanent sutures. These sutures are then placed in the apical ipsilateral edge of the rectovaginal septum and tied down for completion of a bilateral uterosacral colpopexy. This critical and necessary step in the procedure suspends the rectovaginal septum to the uterosacral ligaments, reestablishing the suspensory axis of the vagina and a normal anatomical relationship that was disrupted by childbirth. This surgical maneuver also corrects perineal descent by elevating the perineal body apically into

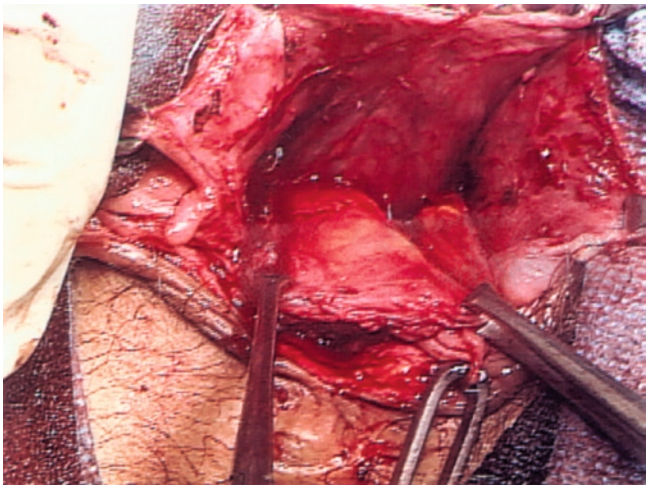

Fig. 18.4 Rectovaginal septum identified

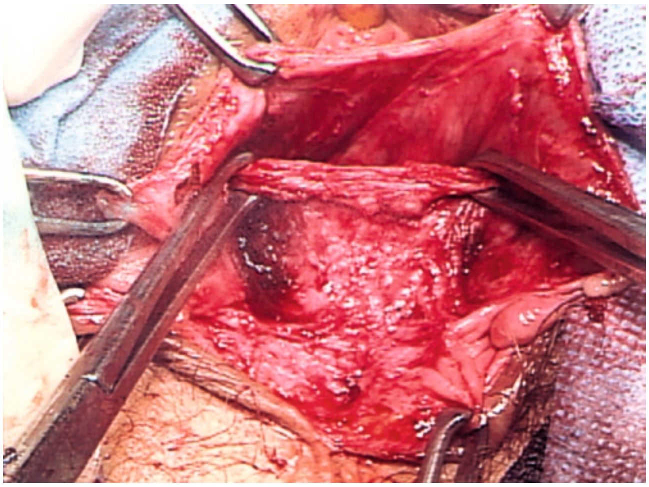

Fig. 18.5 Rectovaginal septum elevated to its original position in preparation for surgical restoration

its appropriate anatomic position within the gluteal fold between the ischial tuberosities. Five permanent sutures make up the complete suspension of the rectovaginal septum. The two lateral sutures attach the rectovaginal septum to the uterosacral ligaments bilaterally, and the three sutures connect the central portion of the rectovaginal septum and the cervix or hysterectomy scar. One of these sutures is placed in the midline and the other two are placed equidistant between the midline suture and the uterosacral colpopexy sutures (Fig. 18.6).

Attention can now be turned o the correction of enterocele and rectocele. Both of these defects can result from the same apical transverse separation of the rectovaginal septum from the pericervical ring resulting in retraction of the rectovaginal septum distally toward the perineal body. This type of defect allows herniation of intestine along with the lining of the peritoneal cavity through the posterior cul-de-sac and rectum through the posterior vagina. Transverse apical defect repair is accomplished by placing three permanent sutures to connect the central portion of the rectovaginal septum to the pericervical ring or hysterectomy scar. One of these sutures is placed in the midline, and the other two are placed equidistant between the midline suture and the uterosacral colpopexy sutures. When these are tied, even a large enterocele and/or rectocele will be successfully and completely reduced without the use of anatomically distorting plication. Correction of the fascial defect repairs the enterocele adequately; there is no need to open the enterocele sac, enter the peritoneal cavity, and risk enterotomy and other complications. Difficulty may be encountered in attempting to sculpt the rectovaginal septum to its original length, especially in patients who have significant obstetric scarring or those individuals who have had previous pelvic reconstructive surgery. Usually, careful release of all lateral and distal secondary adhesions and scars will allow the septum to be surgically sculpted allowing it to reach the interspinous diameter and uterosacral ligaments without excessive tension. If this cannot be accomplished, a bolster of graft should be considered (Fig. 18.7).

If mesh or graft is used in the posterior compartment, familiarity with the bolster of choice and its possible complications along with informed consent for the patient is essential. If artificial material is used, copious and frequent irrigation during the surgical procedure, coupled with anatomic placement, helps to prevent the complications of rejection, exposure, and erosion. Direct attachment of bolsters to the vaginal epithelium should be avoided with use of the endopelvic connective tissue elements instead. The use of non-native grafts and bolsters should be minimized in pelvic reconstructive surgery unless a specific need exists. As previously discussed, the FDA has released guidelines for the placement of mesh in the vagina. Patients must be consented appropriately to both the success rates and possible complications.

If perineorrhaphy is necessary, the perineum should be reconstructed to a width of approximately 4 cm between the vaginal opening and the rectum. Absorbable suture should be used for this portion of the procedure. Artificially narrowing the introitus or plication of the puborectalis muscles should be avoided as this surgical maneuver could lead to dyspareunia. The goal of this portion of the surgery is anatomic restoration of the perineum and not overcorrection. If the fascial sheath surrounding and investing the external anal sphincter needs repair, permanent or delayed absorbable suture should be used. A detailed description of anal sphincteroplasty is beyond the scope of this chapter; however, integrity of the anal sphincter is important to the total integrity of the posterior vaginal segment.

Prospective, randomized clinical trials with long-term follow-up are needed in the area of posterior vaginal

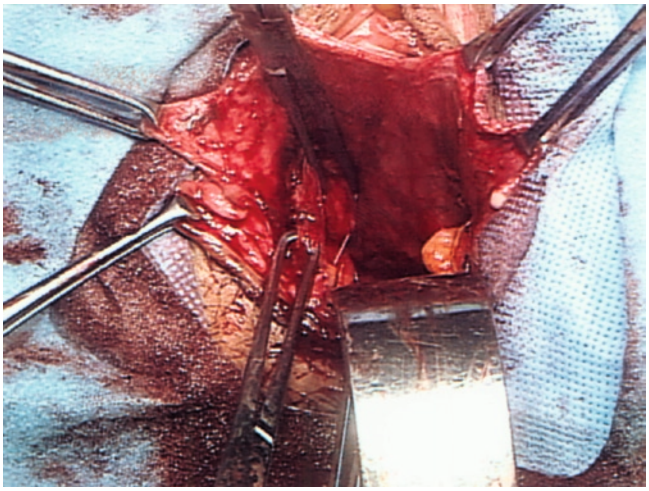

Fig. 18.6 Uterosacral ligament grasped with a long allis clamp

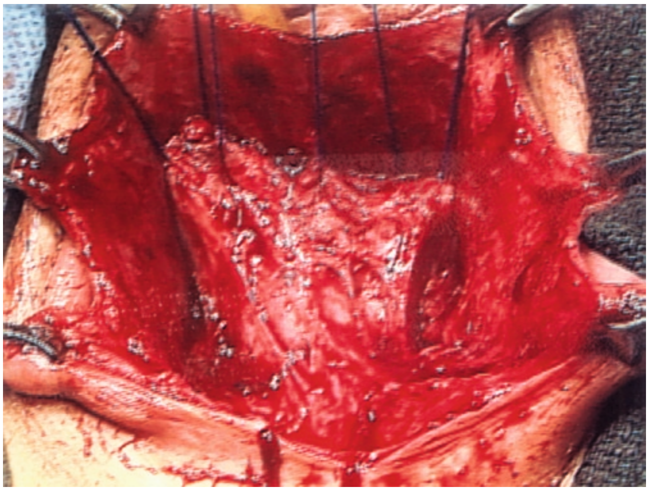

Fig. 18.7 Rectovaginal septum with five apical sutures

reconstruction. With the available information and clinical experience of the authors, the correction of posterior vaginal defects should begin with a suspensory technique such as the uterosacral ligament fixation followed by reapproximation of transverse apical defects of the rectovaginal septum for correction of rectocele and enterocele with permanent suture. This process will adequately address the apical support of the posterior vagina. Continuing distally and laterally with site-specific repair of the rectovaginal septum addresses the mid and lateral support of the posterior vagina. Finally, perineorrhaphy, if needed, should be accomplished for a full-length and full-width repair of the posterior vaginal compartment.

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Rectal Intussusception: Can Posterior IVS Be the Cure?

19

Burghard J. Abendstein

Rectal intussusception is defined as occult rectal prolapse; the prolapsed rectum does not protrude through the anus. Rectal intussusception has been found in 33% of patients with rectoceles and defecatory dysfunction.[1] Typical symptoms are difficulties to evacuate, incomplete evacuation, assisted digitation to aid defecation, fecal incontinence, constipation, impression of a pelvic mass, pelvic pain, and dyspareunia. Endorectal, transvaginal, transperineal, abdominal, or combined approaches are treatment options discussed for symptomatic rectoceles.

Understanding the anatomical basis for rectocele formation is fundamental to planning surgical repair thereof. Nichols analyzed rectocele causation site-specifically[2] and differentiated between a true perineal body defect, and several types of rectocele. Low rectocele was usually caused by dislocation of the rectovaginal fascia from perineal body; mid rectocele by overstretching of the connective tissues between vagina and rectum; high rectocele by damage to the anterolateral attachments of the vagina and cardinal ligaments. Nichols described a fascial attachment between the rectovaginal (Denonvillier's) fascia and levator plate.

A different approach to rectocele repair has been influenced by the Integral Theory.[3] The theory first published in 1990 interprets the anatomy in a dynamic way. The theory states that abnormal bladder symptoms, abnormal bowel symptoms, and vaginal prolapse are related, and are mainly caused by connective tissue defects in three zones of the vagina; lax connective tissue structures invalidate the muscle forces involved in opening and closure of the urethra and anorectum, leading to incontinence (abnormal closure) or retention (abnormal opening). Each zone has three main connective tissue structures. Laxity therein may cause prolapse or abnormal symptoms. Using mesh tapes to reinforce damaged ligaments, up to 80% cure rate was achieved for prolapse, abnormal bladder symptoms, and pelvic pain.[4]

The Biomechanics of Posterior Zone Connective Tissue Damage

As demonstrated in a previous study,[5] it is evident that the PB and uterosacral ligaments (USL) are the anchoring points for the stretching of vagina by the backward/downward vectors. Like a rope suspension bridge, these structures suspend the posterior vaginal wall and anterior wall of the rectum and all are tensioned by the muscle forces. The vagina is lengthened significantly during straining. This "stretchability" derives from the microarchitecture of its collagen and elastin fibers. These fibers are arranged so that no matter which direction the structure is pulled, the fibers become aligned in that direction.[6,7] An elastic fiber network serves as an energy-storing device to maintain the form of the organs.

Elastin diminishes with age and may be damaged at childbirth, whereupon the collagen fibers "droop" under the influence of gravity. Connective tissue in the area of the urogenital organs is sensitive to hormones. During pregnancy, collagen is depolymerized and weakened by placental hormones,[8] allowing dilatation of the birth canal during delivery. Overdistension of the vagina (circles, Fig. 19.1) may cause overstretching of the uterosacral ligaments, posterior vagina, rectal wall, and perineal body. These may rupture (rectocele) or "set" in an extended state after delivery. This process is exacerbated by rapid loss of elasticity with age.

The Biomechanics of Posterior Zone Connective Tissue Repair

The PB and uterosacral ligaments are at least six times as strong as the vaginal or rectal mucosa which they support.[7] The perineal body occupies 50% of the posterior vaginal wall. It is highly unlikely that the fetal head, as it descends down the vaginal canal (circles, Fig. 19.1), will only damage the vaginal or rectal wall in isolation. Damage to USL and PB (Fig. 19.1) is also likely. The PB is a key insertion point of the muscle vectors, as is the USL. Digitally anchoring a lax perineal body under ultrasound control was shown to

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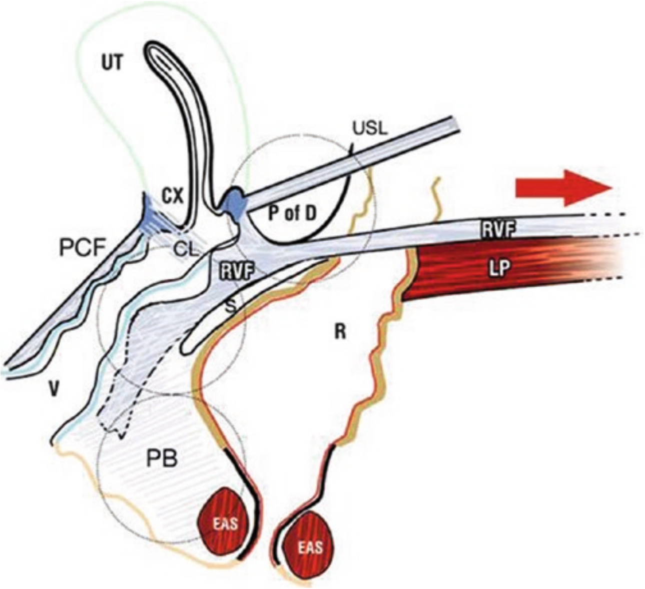

Fig. 19.1 The dynamic structural supports of the posterior vaginal wall – schematic sagittal view. Like a rope suspension bridge, the vagina (*V*) and rectum (*R*) are effectively suspended between the perineal body (*PB*) and uterosacral ligaments (*USL*). The muscle forces (*arrow*) impart strength to this system by stretching it into a semirigid structure. PB is anchored by contraction of the external anal sphincters (*EAS*) and the perineal muscles such as bulbocavernosus (not shown). The rectovaginal fascia (*RVF*) is attached to the USL and cardinal ligaments (*CL*). The rectovaginal space (*S*) allows independent movement of the vagina and rectum (*R*). The *circles* represent site-specific connective tissue damage caused by the fetal head distending the vaginal cavity. *CX* cervix, *UT* uterus, *PCF* pubocervical fascia, P of D pouch of Douglas

alter the geometry and direction of levator plate contraction. This confirmed the role of the perineal body in the active structural-support mechanism. Therefore, it is mechanically not sound to repair the weak vaginal mucosa and not address its structural supports, USL and PB. Simple suturing of a weak USL has never been found to be effective, and so the use of a posterior polypropylene sling has been recommended. On transperineal ultrasound, independent movement of vagina and rectum has been observed, and both organs moved differently from each other during effort. This movement is facilitated by the rectovaginal space (Fig. 19.1). To preserve the rectovaginal space, vaginal tissue should be conserved where possible, and the fascial layers of rectum and vagina should be separately restored. This is especially important if mesh is to be used, as adhesion of mesh to bare rectal mucosa may cause severe dyspareunia or even fistula. Excision of vaginal tissue may obliterate the rectovaginal space and shorten and narrow the vagina, predisposing to future prolapse, as a short vagina cannot be adequately angulated backwards and downwards around the perineal body. Shrinkage of scar tissue around mesh implantation may create such problems in the future in patients with apparently successful operations.

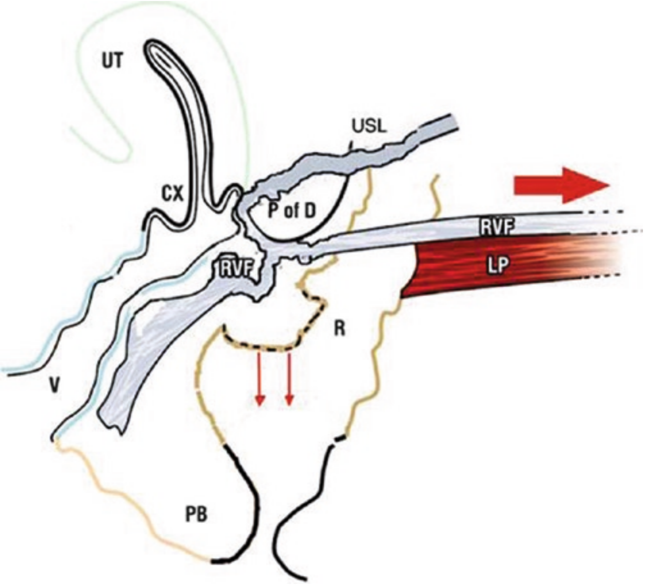

Fig. 19.2 Rectal intussusception. With lax uterosacral ligaments (*USL*), levator plate (*LP*) cannot tension the rectovaginal fascia (*RVF*). The force of gravity (*small arrows*) causes the rectal wall (*R*) to prolapse inwards, much like a tent whose apex is not firmly attached to the pole, "rectal intussusceptions"

Improvement in distended hemorrhoidal veins has been regularly observed after three-level posterior vaginal wall repair. Laxity in the fascial and ligamentous supports of the rectum will cause laxity in the rectal wall (Fig. 19.2) and therefore prevent venous return in the hemorrhoidal vessels. Figure 19.2 demonstrates the importance of competent uterosacral ligaments for support of the anterior rectal wall, much in the way a firmly attached apex supports a tent. Lax uterosacral ligaments (Fig. 19.2) may predispose to both apical prolapse and anterior anorectal wall intussusception. A plastic sling utilizes the negative qualities of foreign-body reaction to create an artificial collagenous neoligament.[9] It can also "reglue" organs and fascia to both muscle and pelvic bone, as demonstrated in the original experimental animal studies.[9] Reattachment of the anterior rectal wall fascia to the uterosacral ligaments can be attributed to the "regluing" facility[9] of this surgical method.

In the presence of rectal intussusception, open or laparoscopic rectopexy, with or without sigmoid resection, is still most widely accepted. Although the anatomic results are mostly good, all procedures widely lack functional improvement. This is in particular true for posterior colporrhaphy,[10] abdominal sacrocolpopexy,[11,12] and rectopexy,[13] all resulting in increasing defecatory dysfunctions. In the normal pelvis, the sacrouterine ligament functions as the most important supporting structure for the uterus, vaginal apex, and via the rectovaginal fascia, also for the posterior vaginal wall and rectum (Fig. 19.1). The rectovaginal fascia (RVF) attaches to the perineal body (PB) below and levator plate (LP) above.[14]

The levator plate is attached to the posterior wall of the rectum. Contraction of the levator plate (LP) stretches both walls of the rectum during anorectal closure and defecation.

In cases with disrupted rectovaginal fascia, a rectocele may form. Due to distended sacrouterine ligaments, the rectum can no longer be kept in its normal position, and consequently, proximal rectal parts may bulge into the distal rectum causing intussusception (Fig. 19.2). According to the Integral Theory,[15] dysfunctions of anorectal opening (evacuation disorders) and closure (fecal incontinence) are mainly caused by connective tissue damage in the vagina or its suspensory ligaments. The explanations offered above expand these concepts to the pathogenesis of rectal intusssusception. The infracoccygeal sacropexy ("posterior IVS")[16] procedure belongs to the family of "tension free tape" operations. An implanted polypropylene tape (Tyco Healthcare) reinforces the uterosacral ligaments by irritating the tissues to create a linear deposition of collagen. In contrast to other methods which aim at fixation of the rectum, posterior IVS does not attach the vagina or the rectum firmly to bony structures. It allows the surgeon to restore the normal vaginal axis and the rectovaginal fascia anatomically correctly, thereby reestablishing normal function.

The rationale for the use of posterior IVS in order to treat symptomatic rectoceles with intussusception is founded on three main reasons:

1. Baden and Walker pronounced their tent theory,[17] stating that if the top of a tent caves in, the walls may follow. This translates for the vaginal situation that the first step in the treatment of vaginal or even rectal prolapse should be the restoration of a competent apical fixation, namely restoration of the sacrouterine ligaments by insertion of a polypropylene tape (posterior IVS). Important in this type of anatomical restoration is buttressing of the side walls, namely the rectovaginal fascia in cases of a rectocele.[16]
2. The technique of posterior IVS follows the Integral Theory surgical principles, that "restoration of function follows restoration of form."[18] As a consequence, it seems obvious that restoring the ligamentous supports of the organs is more promising than other methods that work by stretching the organ and attaching it to fixed structures, either the rectum (rectopexy) or the vagina (sacrocolpopexy).[11-13]
3. Prior personal surgical experience with the Posterior IVS operation (PIVS) in patients who had prolapse, and who were also cured of their defecatory problems, suggested that this principle could be widely applied in patients with symptomatic rectocele and rectal intussusception.

In a prospective trial, we could demonstrate that rectal intussusception can be cured by reconstructing the posterior zone anatomy, uterosacral ligaments (posterior IVS), rectovaginal fascia, and perineal body.[19] Forty-eight patients with various degrees of vaginal vault descensus, clinical rectoceles with defecatory dysfunctions, and documented rectal intussusception (proctography, Fig. 19.3) were treated by insertion of a posterior IVS, reconstruction of the rectovaginal fascia, and perineal body repair. Of the 48 patients with evacuation difficulties, 45 (94%) patients reported complete normalization of defecation at both visits after surgery. Of the 27 patients with fecal incontinence, 18 (66%) reported cure, 5 (19%) >50% improvement, and 4 no change. Postoperative proctograms (Fig. 19.4) showed resolution of the rectal intussusception in all cases and all

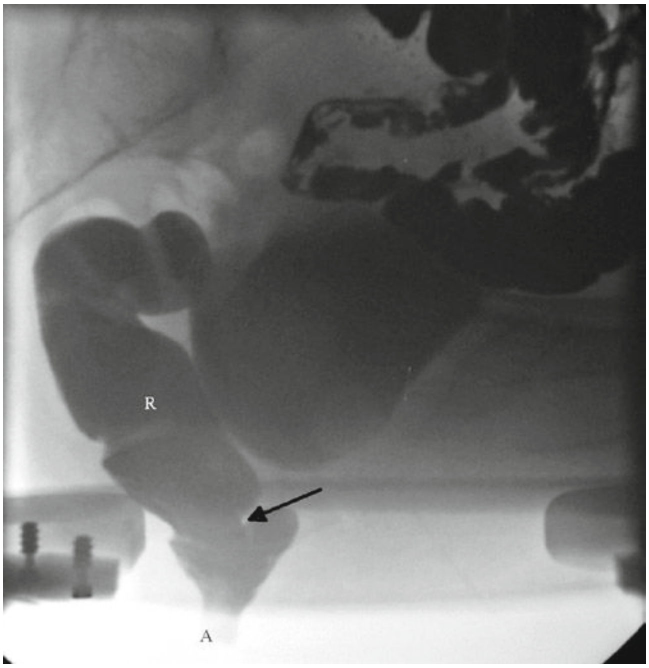

Fig. 19.3 Preoperative defecating proctogram. Sagittal view, straining. *Arrow* indicates site of intussusception on anterior wall of rectum (*R*) which is misshapen and is obstructing evacuation; *A* anus

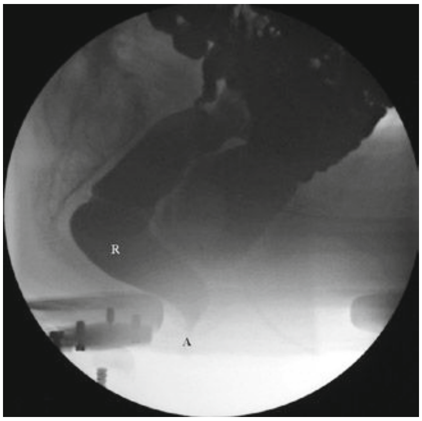

Fig. 19.4 Postoperative defecating proctogram. Sagittal view, straining. The intussusception has disappeared. The rectum (*R*) has a normal shape, and evacuation is proceeding normally through the anus (*A*)

patients reported completely normal defecation after surgery. In this study, only minor complications occurred, such as little erosions (4%) easy to treat by local excision. One single rectal perforation was detected at primary surgery and could be managed uneventfully by primary suture.

These results appear to confirm the hypothesis that the sacrouterine ligaments are an essential structure for normal function of the anorectal complex.[15] Furthermore, reinforcement of the sacrouterine ligaments by insertion of a posterior IVS tape is successful in restoring both anatomy and function. This "tension-free" approach sets out to mimic normal anatomy without distortion, by repairing all the anatomical levels which contribute to anorectal opening and closure.[16,18] This three level approach does not alter the geometry or the axis of the pelvic organs, and would appear to offer a more anatomical, and therefore, more functional treatment plan than isolated rectopexy or sacrocolpopexy. The latter perform only a level 1 repair.

In order to achieve best functional results stretching and overcorrection of the organs should be avoided. This is certainly the case for rectopexies. During rectopexy, a distance of about 10 cm of the rectum is functionally disturbed by fixation of the rectal wall onto the sacral promontory. This may be the reason for the reported poor functional results of this procedure. The same reservations apply for sacrocolpopexy, a method which bears the problem of overcorrection if fixated to the sacral promontory. Using posterior IVS avoids the danger of overcorrection, since neither the rectum nor the vagina is fixed to bony (and therefore immobile) structures during this procedure. Above all, organ mobility, a key element in pelvic floor function, can be maintained, as described in the Integral Theory.[15-18,20,21]

Compared to rectopexy, posterior IVS is less invasive and thereby less susceptible to surgical complications. In a large series,[22] 33% operative morbidity was reported after rectopexy. Most complications occurred in the early postoperative period, including severe complications like bowel obstruction and ileus, but also late complications and fistulas occurred. In our series, we did not have a single severe complication. Erosions (4% incidence) and their accompanying vaginal discharge may sometimes be disturbing to the patient, but they are a minor problem, and usually easy to treat.

Based on the results of our study, we integrated posterior IVS technique in routine clinical practice for the treatment of intussusception and obstructed defecation with continuing success.

Summary

With regard to "obstructed defecation" and rectal intussusception, the use of posterior IVS offers clear clinical advantages compared to more conventional procedures, minimal pain and trauma, rapid recovery, and fewer complications. It is a correct anatomical approach with no unphysiologic fixation of the rectum. Thereby, the function of defecation is restored and there is a high chance of rapid normalization of stool habits immediately after surgery. Keeping to the surgical principals of posterior IVS technique allows the combination of infracoccygeal slings with pieces of mesh for restoration of the rectovaginal fascia or the use of modern precut mesh products without losing the desired outcome effects.

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Part VI

Complications

Exposure and Erosion of Vaginal Meshes: Etiology and Treatment

20

Carl W. Zimmerman, Peter von Theobald, and Naama Marcus Braun

Introduction

In various parts of the body, surgeons use several types of materials to increase the durability and strength of hernia repairs. For that reason, grafts have become commonplace in pelvic organ prolapse surgery. Much of the data used to justify this approach have an origin in the ventral hernia repair literature. Because of the documentation of better outcomes of hernia repairs on the abdominal wall when grafts are used, many products have become commercially available for use in pelvic reconstructive surgery. While definitive data are lacking in most areas of gynecological use, evidence-based benefits have been demonstrated in urinary slings and abdominal sacral colpopexy. A lucrative manufacturer-driven industry has arisen that has led to the availability of a wide array of kits that allow implantation of various types of synthetic thermoplastic polymers, such as polypropylene, allografts, and both cross-linked and noncross-linked xenografts. These implants and permanent sutures, both multifilament and monofilament, may cause postoperative problems. In theory, implantation kits can potentially help to compensate for the inherent connective tissue weaknesses encountered during pelvic organ prolapse surgery. The inherent challenge of prolapse surgery resides in the ultimate goal of suspending the vagina, which is surrounded by functional gastrointestinal and urinary organs, over an opening in the pelvic floor large enough for a term infant to pass through. Furthermore, the native connective tissue involved in this type of repair has been subjected to significant physical stress during childbirth, has avulsed from normal anatomical relationships, and has been displaced away from the interspinous diameter and the normal attachments to the pelvic sidewall.

Significant differences exist in the surgical environments of the abdominal wall and the vaginal vault. Some of the salient contrasts are listed in Table 20.1. In the abdominal wall, the buffering dermal, fatty, and musculofascial layers help to protect implanted materials from exposure to the epithelium and its resident bacterial population. No such buffering layers exist between implanted materials and the epithelium within the vagina. Direct contact of the vaginal epithelium with bolsters predisposes these materials to both early and late failure of complete healing. Compounding this physical proximity is the fact that vaginal incisions are classified as clean-contaminated because of the plethora of microorganisms that are present in the normal vagina. On the other hand, ample reasons exist that help to justify the use of bolsters in the repair of prolapse. For example, the endopelvic connective tissue margins that must be repaired in site-specific pelvic reconstructive surgery are damaged as described in the previous paragraph. A stronger repair is certainly a worthy goal in this circumstance. In the ventral abdominal wall, a hernia does not occur through a functional opening like the vagina, nor is it surrounded by functional organs like the bladder and rectum. The three-dimensional anatomical complexity of the intact endopelvic fascia coupled with the functional sensitivity of the vagina and its surrounding structures makes any problem associated with implanted material more likely to be clinically obvious to the patient and surgeon.

At the present time, no definitive data exist regarding how much implanted material should be used in prolapse surgery, when it should be used, or if the benefits of bolsters outweigh their risks over an extended period of time. Three concepts related to this concept seem logical. If a bolster is to be implanted, it should have a biomechanical function that at least theoretically provides a benefit to the strength of the repair. In addition, no more mesh than necessary should be used in order to accomplish the stated goal of a durable repair. Finally, if a material is implanted for a non-lifesaving quality of life surgery, for example, prolapse repair, the surgeon should be able to remove all or a substantial portion of it in the event of a significant complication or functional impairment that is due to the implant. Commercial kits with large areas of mesh and multiple insertion arms that are marketed as substitutes for meticulous reconstructive surgical techniques do not necessarily satisfy these criteria. Data that

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Table 20.1 Location differences

Abdomen	Vagina
Mesh is placed deep and not in direct contact with the skin	Mesh is placed adjacent to the epithelium
Theoretically sterile insertion area	Very high bacterial colony counts
Surrounding connective tissue is undamaged	Childbirth damage to all endopelvic connective tissue
Structurally simple hernias	Very complex hernias with gi, gu, and sexual functions as variables

show improved anatomical and functional outcomes would be required to justify implantation of these devices. Pelvic surgeons should use implants carefully and judiciously in order to avoid exposure, erosion, infection, and scar contraction of the implanted material.

Prevention of Complications

The best treatment for any complication including exposure and erosion of vaginal mesh materials, mesh-related pain syndromes, functional impairment of the patient, and failure of prolapse surgery is prevention. Techniques for prevention of problems with healing of an implanted material include precise surgical techniques, e.g., good hemostasis, dissection in the correct avascular plane, attachment of the bolster to the correct support and suspensory structures, and irrigation of the surgical field throughout the case. Careful use of electrocautery and sutures helps to establish hemostasis and prevent hematomas. Precise dissection is necessary to help identify the avascular spaces of the perivaginal area that are necessary to gain access to key support and suspensory structures. Success with both of these surgical skills is helpful in avoiding poor healing. The vagina is surrounded by a plexus of veins that are directly adjacent to the vesicovaginal and rectovaginal spaces. Some surgeons also use postoperative packing to tapenade oozing in the immediate postoperative period of time. Other pelvic surgeons use drainage to avoid hematomas that might become contaminated in this semi-aseptic area.

Irrigation of surgical fields has been shown to prevent postoperative infection in the orthopedic and in the abdominal surgery literature.[1-4] However, in the abdominal surgery literature where surgery typically occurs in a clean field, there is now a debate, because irrigation dilutes peritoneal immunologic reaction as well as microorganisms. Gynecologic surgeons routinely use irrigation during abdominal and laparoscopic procedures for dilution and reduction of bacterial contamination; however, this practice is not so widely used in vaginal surgery. In an operative field that is laden with a diverse variety of microorganisms and is the site of foreign material implantation, irrigation of the operative site throughout a pelvic support case seems to be a logical maneuver. Devices exist that allow for ergonomic suction, irrigation, and lighting. These capabilities can assist in the performance of surgery and prevention of complications (VersaLight, Lumitex, Inc., Strongsville, OH; VitalVue, ValleyLab, Boulder, CO).

The avascular spaces of the pelvis are a primary resource for successful pelvic surgery regardless of operative approach (see Chap. 1). For biomechanically sound pelvic reconstructive surgery and the identification of key anatomic structures in the deep pelvis, the vaginal surgeon relies on the vesicovaginal, rectovaginal, paravesical, and pararectal spaces for access to key support and suspensory structures. These potential spaces need be successfully dissected in order to avoid excessive intraoperative blood loss, hematoma formation, and subsequent problems with infection. Any of these complications predispose to poor healing and mesh exposure. Successful apical suspension to the uterosacral ligaments, sacrospinous ligaments, or other acceptable suspension structure is dependent on isolation of these structures by deep dissection through these spaces. Likewise, the arcus tendineus fascia pelvis used for midvaginal lateral support is best accessed through the vesicovaginal and rectovaginal surgical planes that extend to the pelvic sidewall denoted by the obturator internus muscle. If mesh is attached to these or other key biomechanical structures in a surgically precise way, exposure and erosion, bunching of the mesh, pain syndromes, and failure of the surgery are less likely to occur.

When possible, care should be taken to avoid implantation of allograft and nonremodeling xenograft and allograft materials directly under vaginal incisions. The absence of buffering layers allows for direct contact to the epithelium of the vagina and any break in the incision during the healing process will predispose to faulty healing. Remodeling xenografts do not seem to share this characteristic and may be placed directly under an incision or used to help exposures and erosion heal. Use of the avascular spaces affords the surgeon the opportunity to dissect under the epithelium and strategically place grafts away from direct contact with incisions.

Treatment of Complications

A summary of potential complications from implanted materials within the vagina and pelvis is given in Table 20.2. Each of these problems is discussed in turn.

Exposure and erosion of implanted materials is the most common problem encountered in implanted prolapse patients. Pelvic reconstructive surgeons are just beginning to define

Table 20.2 Complications associated with implanted materials in pelvic reconstructive surgery

Exposure
Erosion
Infection
Granuloma
Seroma (cyst formation)
Scarring
Pain
Fistulae

the extent of these problems and publish how these complications can be adequately addressed.[5,6] The reported incidence of failure of vaginal epithelial healing is not known with precision for various reasons. Reports of this complication vary widely in the literature. Long-term follow-up is not common in these series. In addition, most pelvic surgeons who see a large number of these complications are located within referral centers. In this circumstance, a selection bias exists for the more severe cases to the exclusion of problems that are successfully managed in a more conservative way or that never come to the reporting center. In other words, the denominator of the equation (total number of implants) is better known, than the magnitude of the numerator (total number of erosions and exposures). This concept of likely significant underreporting is applicable to other implant-related complications as well.

Some aspects of exposure and erosion are clear. When a foreign object is implanted within the vaginal microenvironment, exposure, erosion, and painful granuloma will occur in a clinically noteworthy percentage of the cases.[7-9] The degree of morbidity for the patient will vary from case to case. Some of these complications may be treated conservatively in the office setting, while others require operative intervention. In a small percentage of the total number, major surgery will be required and, even with that intervention; the patient's perception of her symptoms may persist despite the surgeon's best effort at explantation or revision.

Permanent suture exposure with a granulation reaction can usually be treated by simple excision in the office setting. Often, a bud of red friable granulation is the clue to an embedded permanent or delayed-absorbable suture or a portion of an implant. Postcoital or activity-related vaginal bleeding is the usual presenting complaint. Prior to removal of the suture, pretreatment with an analgesic may be required if the patient is unable to tolerate the manipulation required to search for the offending material. A long Kelly or Vanderbilt clamp can be inserted into the vagina outside the lateral margin of the speculum in order to leave the central opening of the speculum available for visibility and for insertion of a long curved scissor. Once the suture or material is isolated, it can be grasped with gentle traction and the loop below the knot can be cut to remove the entire suture or to trim the implant. If sufficiently large, a granulation bud or polyp may be trimmed or cauterized. Follow-up at subsequent visits can assure resolution of the granuloma reaction. If bleeding or a granuloma reaction persists, a more complete exploration may be required especially if the permanent suture was attached to a mesh or nonremodeling bolster at the time of the initial surgery. In those cases, a more complete dissection and wider excision may be necessary in the operating theater with adequate anesthesia and instrumentation (Fig. 20.1).

Synthetic fiber mesh and both allograft and xenograft bolster exposure and erosion vary greatly in clinical severity depending on the type and amount of material that was initially implanted and the degree of development of the problem. These complications may be asymptomatic, and if so, they do not require treatment unless the patient expresses a desire for intervention (Fig. 20.2). Common presenting complaints include abnormal discharge, spontaneous and postcoital bleeding, female dyspareunia, male complaints during coitus, and pain syndromes. Cross-linked xenograft implants may create a nonhealing and painful granuloma reaction that can cover a large area of vaginal epithelium and tends to be persistent despite conservative measures. These implants cannot be remodeled by the biochemical mechanisms of the body. Excision of the affected epithelium, the underlying graft, and surrounding inflammatory reaction may be necessary to alleviate the symptoms. Usually, the margins of the graft can be visually identified or palpated (Fig. 20.3). These landmarks can be successfully used to complete excision and to avoid removing any more tissue than necessary and injury to underlying structures. Seroma formation may be encountered with allograft and xenograft materials that are not

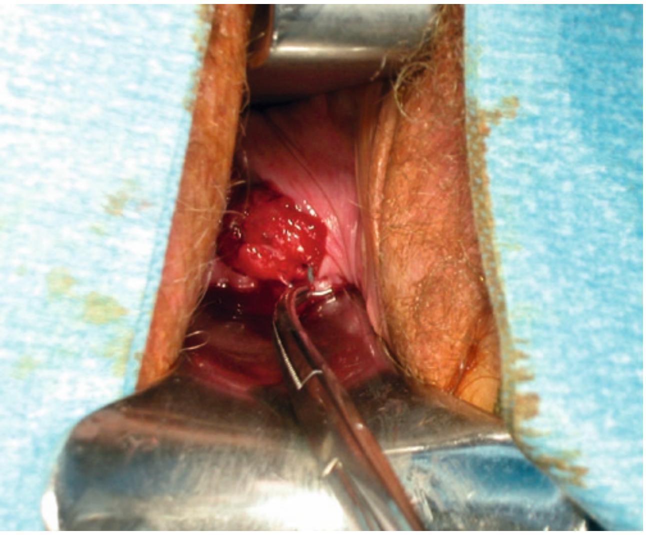

Fig. 20.1 Permanent suture granuloma following apical vaginal reconstruction

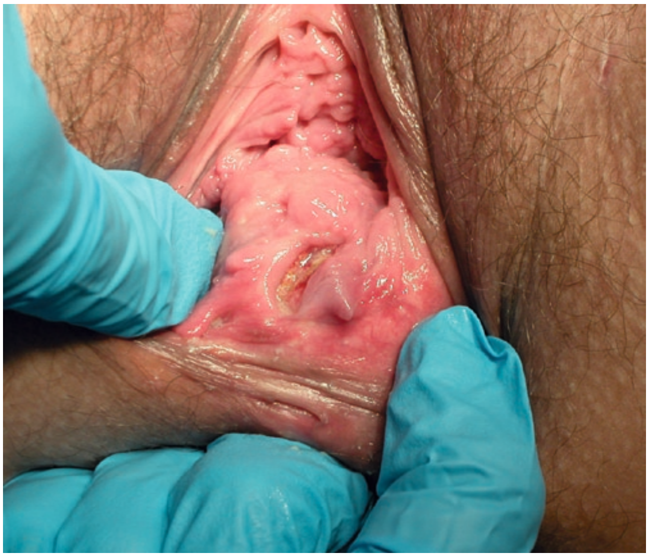

Fig. 20.2 Painful exposure of permanent polypropylene mesh in the distal posterior vagina that required surgical excision

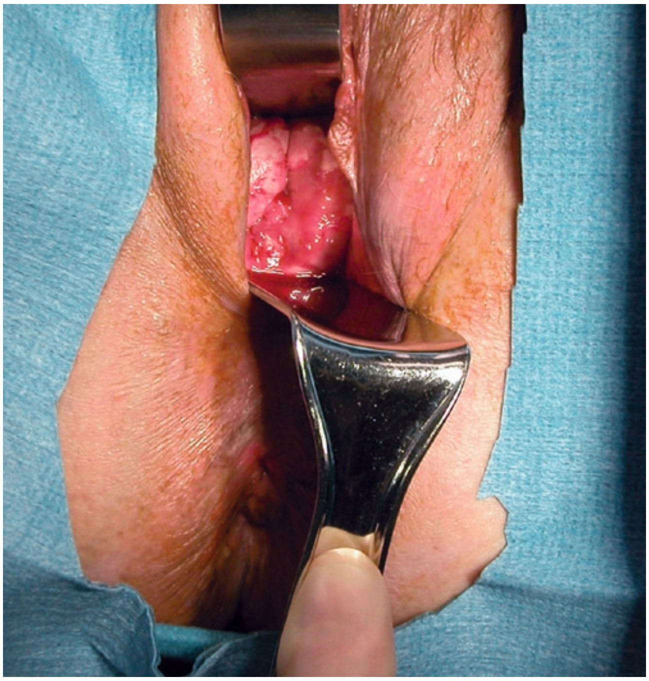

Fig. 20.3 Chronic inflammatory granuloma after implantation of cross-linked porcine xenograft

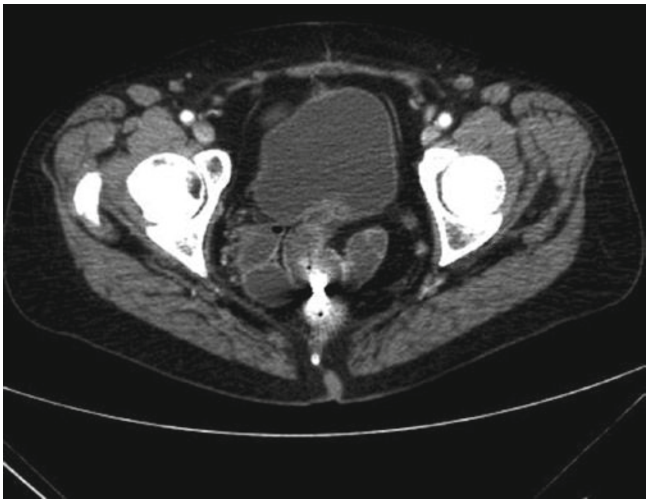

Fig. 20.4 CT of seroma formation after use of cross-linked porcine dermis material in pelvic reconstructive surgery

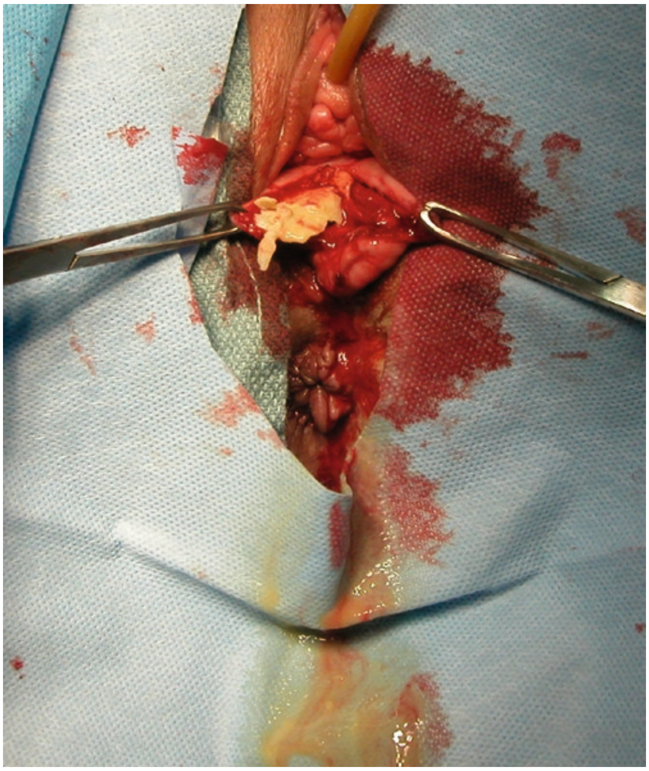

Fig. 20.5 Surgical intervention for seroma seen on CT in Fig. 20.4. Note the persistence of graft material within the seroma cavity

incorporated into the connective tissues of the host. Incision, drainage, irrigation of the cavity, and removal of the foreign material are required for treatment (Figs. 20.4 and 20.5). A neovascular reaction induced by the body's attempted healing mechanism and associated inflammation can be a hemostatic challenge when operating on graft and granuloma excisions. For that reason, fine dissection with a Colorado micro dissection tip cautery (Stryker, Kalamazoo, MI) is helpful with a low energy setting in the 30 W range. This technique seals capillary and other small vessels as the dissection progresses and does not interfere with visual identification of specific tissues or palpation of graft and margins of inflammatory reaction. Frequently, the bowel or bladder must be closely approached in the process of graft excision. The microsurgical control and hemostasis offered by this precise electrosurgical technique are superior to traditional macrosurgical techniques. After the affected area is excised, closure of the epithelium should be accomplished primarily if possible, especially if only the anterior or posterior wall of the vagina is affected by the clinical problem. If the surgical defect is too large or is acutely or chronically infected, the incision can be left open to heal by secondary intention and to allow for drainage during the healing process. If both the anterior and posterior walls are affected, and if primary closure cannot be adequately accomplished, steps should be taken to avoid secondary adhesion formation that can create the same vaginal architecture as a Lefort colpocleisis with a midvaginal adhesion occluding the central portion of the vagina. Remodeling bolsters, such as porcine small intestine submucosa, Surgisis Biodesign™ (Cook Biotech, West Lafayette, IN) or bovine dermis, Xenform™ (Boston Scientific Inc., Natick, MA) can be used to safely cover these defects and help to avoid secondary adhesion formation. Loose packing that is changed frequently in the office setting or a neovagina mold can also be effectively used. In all mesh, bolster, suture, and graft excisions, the patient should be informed about the possibility of the need for staged or additional procedures in the future. Many of these problems require scar lysis, additional excision of residual material, or secondary repair of recurrent prolapse at a subsequent time. Outlining the potential scope of the problem at the beginning of treatment can assist the patient in creating realistic expectations for these potentially challenging procedures.

Permanent mesh material is responsible for the majority of cases that require removal or revision of urogynecology or pelvic reconstructive surgery implant material. A learned discussion of the composition, construction, and justification for the use of these materials is covered elsewhere within this book (see Chap. 10). The most commonly used permanent thermoplastic polymer that is used as a mesh graft in prolapse surgery is polypropylene. Several different weaves that have varying size of interstices within the mesh are used. Early meshes often had a multifilament weave with small interstices construction that was speculated to impede tissue ingrowth as with the IVS Tunneller (TYCO, Norwalk, Connecticut). Others believe that the problem wasn't one with ingrowths. Rather, due to the small mesh interstices, a localized devascularization of the vaginal epithelium was induced and the erosion rate was much higher than with other mesh weaves. Suspicion without clinical evidence of higher infection rate exists, as well. Lack of ingrowth was the problem with polytetrafluoroethylene (Gore Tex™, Flagstaff, AZ) implantation within the vagina and the abdomen, and its use was quickly abandoned because of high exposure and erosion rates. Some valid criticisms exist of the use of multifilament polypropylene mesh. One valuable benefit to this type of permanent material is that if excision is required, it can be accomplished in a relatively easy and complete fashion because of the relative isolation of the fabric of the mesh from the surrounding tissue. More recently, manufactures have increased the size of the openings in implantable meshes in order to allow complete tissue integration as the healing process occurs theoretically allowing for greater eventual tissue strength. When primary healing occurs, this tissue ingrowth can be good; however, when exposure and erosion occur, excision of the affected material can be extremely difficult and may require potentially extensive surgical procedures. Usually, partial excision of exposed mesh and some surrounding inflamed tissue is sufficient to relieve the patient's complaints. Once again, microtip cautery (Colorado tip®, Stryker, Kalamazoo, MI) dissection can make this task technically easier and more hemostatic than traditional dissection and electrocautery techniques. The surgical goal with newer polypropylene mesh is usually not complete mesh excision. Once noninflamed margins can be identified, the procedure can be terminated and the patient can be followed for evaluation of complete healing. Original operative notes are helpful in identifying the specific implant that was used and the technique of implantation. If the type of implant is known, the likely maximum anatomical margins of the proposed excision can be deduced. Adequate evaluation of adjacent organs is a valuable adjunct to these surgeries. Cystoscopy to validate ureteral patency and the absence of bladder involvement may be prudent prior to the initiation of extirpative surgery. Likewise, evaluation of the sigmoid colon or rectum may be required. These diagnostic tools may be used before, during, or after any given surgical procedure. If mesh scarring is palpable or a sinus tract is present outside the vagina, radiologic imaging may be helpful to identify abscess, seroma, or fistula formation (Fig. 20.6).

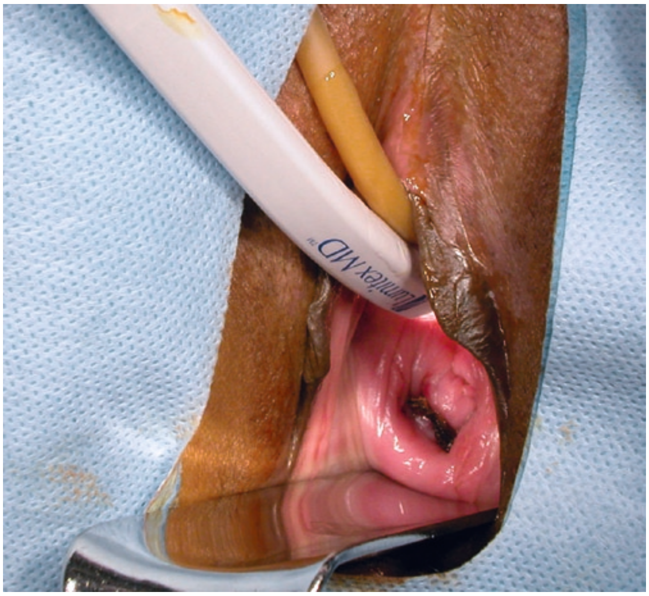

Fig. 20.6 Exposed multifilament polypropylene posterior sling coated with biofilm that required explantation due to chronic bleeding and discharge

Envision a circumstance where a large multiarmed polypropylene mesh is partially exposed and infected with a drug-resistant bacterium, e.g., methicillin-resistant *Staphylococcus aureus*. Removal of the total mesh implant would be in the best interest of the patient's health. In this circumstance, an extensive surgery procedure may be required that could involve the vagina, the adjacent surgical spaces, bladder, rectum, and possibly an abdominal incision if access to the area adjacent to the internal aspect of the obturator membrane is required. Exploration of the ischioanal space via the buttock may also be required. The arms on these products are introduced into the central pelvis through the ischioanal space via the buttock, the paravesical space via the obturator membrane, and through the prevesical space via the urogenital diaphragm. Obviously, if the need arises for a complete excision, surgical challenges abound. Experienced surgeons and proper caution should be used in these circumstances. Multispecialty surgical consultation may be required. At times, large areas of the vaginal epithelium need to be removed to relieve a patient's symptoms. In a patient with continued coital desire, skin grafting, tissue manipulation flaps, and liberal use of non-cross-linked remodeling biomaterials can be helpful.[10,11] If the excision has been extensive, the same protocols used for neovaginal construction may be needed. If the patient has no further coital desires, a vaginectomy along with complete colpocleisis may be needed and may be the only expedient way to relieve the patient's symptomatology (Fig. 20.7).

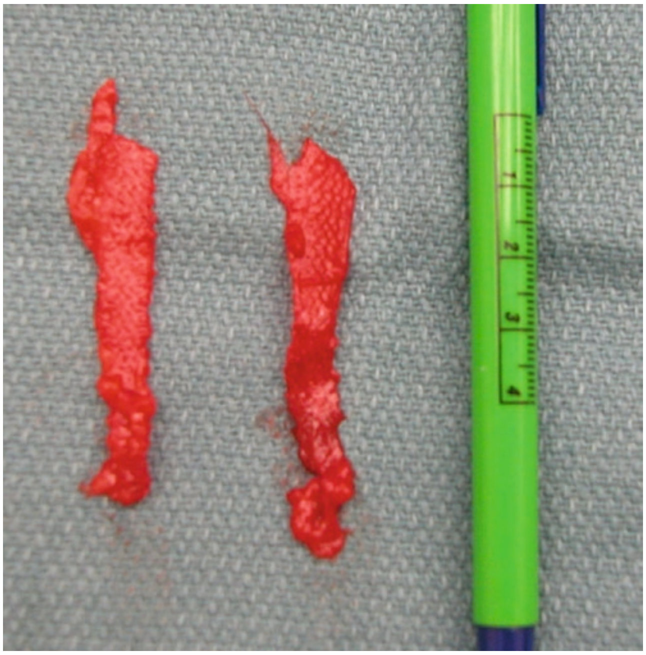

Fig. 20.7 Retropubic suburethral sling excised with vaginal and retropubic dissections because of methicillin-resistant *Staphylococcus aureus* infection

Granuloma formation in the setting of a previously implanted pelvic floor mesh or bolster is almost always a foreign body reaction. For that reason, a persistent and especially painful granulation reaction should be assumed to contain either suture or graft material as the cause. For reasons that are not well understood, granulomas may or may not be painful. Granulomas frequently cause a discharge that may be bloody and often present with postcoital or activity-related bleeding. Time is required for a large granuloma to develop. For that reason, these complications do not present early in the healing process. When detected, many of these issues may be managed in the clinic or office setting; however, if pain is present, operative intervention may be required. Forceps and scissors, a cervical biopsy instrument, and silver nitrate are useful in debulking a granuloma. If a permanent suture is detected, care should be taken to identify the loop end of the suture prior to cutting. If the entire suture is not removed, the process will likely persist. A very large granulomatous polyp can be excised with cautery or encircled with an Endoloop® (Ethicon Endosurgery, Blue Ash, OH).

Seroma formation can occur in the presence of both biomaterials and permanent polymers (Figs. 20.4 and 20.6). These fluid collections do not represent an infectious process, but may reach a significant size. In the process, they may protrude from the introitus, cause pressure symptoms in the bladder or rectal ampulla, cause dyspareunia, or simulate a return of prolapse. Imaging with CT technology is valuable to define the anatomical limits of the fluid-filled cavity. Incision, drainage, and meticulous removal of any foreign materials exposed to the seroma cavity will usually result in resolution of symptoms. Seroma is usually a late presentation complication and insidious in its onset.

Abscess is usually an early presentation complication; however, they may occur months or years after the initial surgery. Pain, spontaneous intermittent drainage, and fever are the triad of presentation. Because of the presence of foreign material, conservative management is unlikely to adequately manage the process. Incision and drainage is required along with excision of any foreign material that is located within or close to the abscess cavity. Frequently, the incision must be left open and healing by secondary intention allowed to occur. Cultures and exclusion of fistulae should be accomplished. If a fistula is present, the gastrointestinal tract is commonly involved in the presence of an abscess. Gastrointestinal diversion may be required consistent with the concept of a staged procedure. Closure of the diversion would be planned after the acute process has resolved.

Subacute rejection of unknown etiology occurs in 1–2% of mesh implantation patients. The presentation may be late, up to 1–10 years after insertion. Symptoms are constant or intermittent discharge and bleeding often with a complaint of odor by the patient. Usually no pain or fever is present. At examination, it is sometimes difficult to find the small vaginal fistula through which the discharge escapes. Repeated examinations are sometimes necessary for identification. A localized swelling in the vaginal wall may be palpated, and if pressure is applied, the production of seropurulent fluid will help identify the opening. Surgical assessment through a vertical colpotomy will often find pus-like liquid, usually negative at microbiologic cultures. The mesh is usually coated with biofilm and may be totally dissected by following this brownish black "slime" with blunt being more productive than sharp dissection. This method of explantation can lead to relatively easy removal of the mesh. Frequently, total removal by gentle traction and meticulous dissection is possible. The epithelium may be closed after disinfection or at a later time. Usually in this circumstance, the prolapse does not recur because of the presence of a fibrous reaction that surrounds the mesh. Debated origins for this type of problem include immunologic rejection or a chronic, slowly dissecting, much localized infection around the mesh. Mesh cultures are almost always negative for bacterial growth.

Scarring is a frequent and troublesome late complication of mesh and bolster placement. It may be very difficult to manage. Especially with larger multiarmed mesh implantations, scarring may significantly narrow the caliber of the vaginal vault and result in various degrees of pelvic pain and dyspareunia. Scarring commonly coexists with other implant complications. When the problem is sufficiently symptomatic, scar contractures should be managed surgically. Conservative measures such as vaginal dilators and repeated massage of the constricted area are unlikely to result in significant relaxation of the contracture. Operative management should be planned in a way to remove all or a significant amount of the implanted material including the cicatrization surrounding it. Careful electrical microdissection is helpful for avoidance of visceral structures. Scar release with a relaxing incision, z-plasty, tissue advancement flap, or other tissue manipulation flaps may be helpful.[10,11] Obviously, the goal in this setting is to restore depth, axis, and caliber to the vaginal vault and to relieve pain. If a large area of scar is present, skin grafting may be required.

Management of significant pain following graft or bolster-assisted pelvic reconstructive surgery is the ultimate challenge of this type of surgery. Obvious causes of discomfort such as nerve entrapment may exist. Sacrospinous ligament fixation and other procedures that use those ligaments for apical suspension are particularly prone to this type of pain. The sciatic, pudendal, levator ani, or inferior gluteal nerves may be involved. Diagnosis is dependent on the distribution of the patient's neurological symptoms. These syndromes often present immediately after surgery and may require immediate operative management by suture removal. Pain may also occur as a result of scar contracture around a polymer or biologic implant. The causation of this pain is uncertain although some cases certainly appear to be a result of infection-related scarring or scar contracture. Excision of scarred and retracted mesh or bolster material may be required. These surgeries are difficult because of the wide areas of vaginal epithelium that may need excision, contracture of the implant with dense tissue ingrowth, and the need to preserve as much normal vaginal anatomy as possible. Pressure on a palpable area of mesh contracture or exposure will allow the surgeon to determine if a particular palpable mesh contracture is involved in a pain syndrome. Other causes of pelvic pain such as pelvic floor spasm, fibromyalgia, or interstitial cystitis may be unmasked or exacerbated by implantation of bolsters of all types (Fig. 20.8).

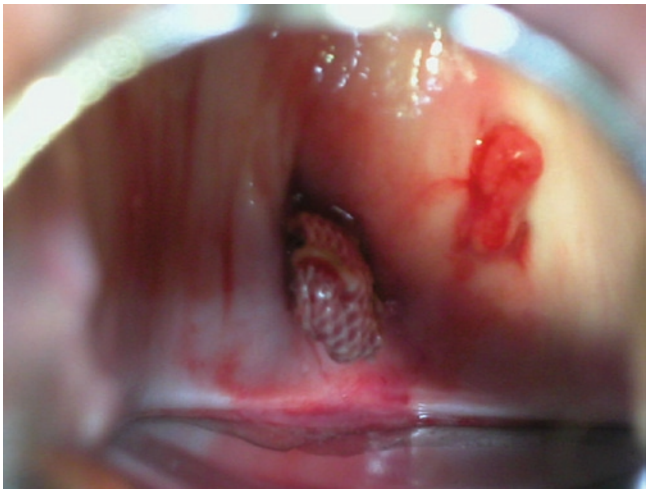

Fig. 20.8 Chronically exposed abdominal proctopexy graft in the apex of the vaginal vault that required excision

Fistulae may occur early or late during the patient's recovery process[12] (Figs. 20.9 and 20.10). Symptoms are directly related to the location of the inflow tract(s) of the abnormal connection. Often these processes are not painful, although abscess may accompany gastrointestinal fistulae. Management requires removal of foreign material and closure of the fistula. Diversion and staged procedures are not uncommon. Surgical management of fistulae is beyond the scope of this chapter. The myriad of potential fistula types and the various techniques that are used in this type of surgery are not discussed. Obviously, if an implanted material has caused a urinary, intestinal, or infectious fistula, surgical correction will be required with excision of the foreign material being necessary to accomplish successful fistula closure.

The gynecologic surgeon may encounter mesh exposures and erosions implanted by other specialties or in adjacent organs. In those circumstances, appropriate consultation and an understanding of the likely location of the materials is critical. Cystoscopy, anoscopy, laparoscopy, and possible laparotomy may be required to address the problem.

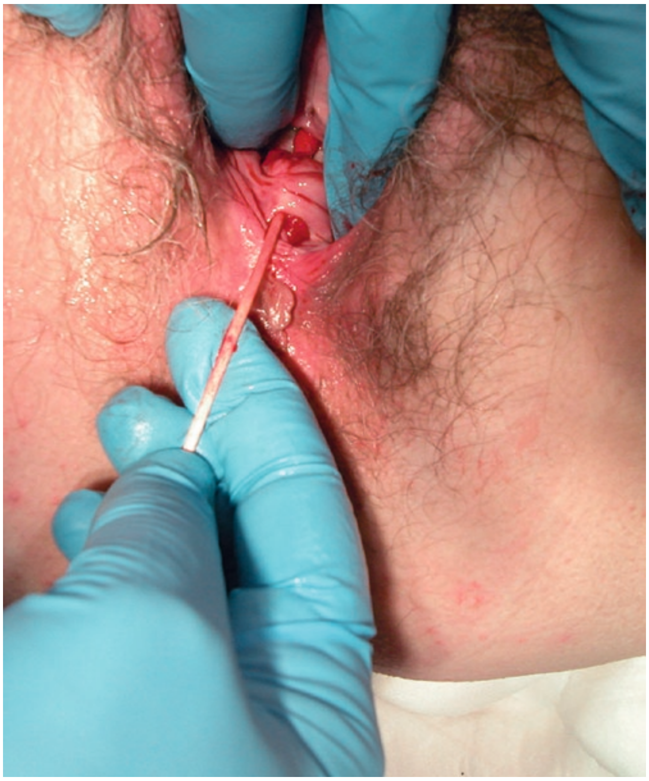

Fig. 20.9 Cutaneocutaneous fistula in the vagina that developed after use of porcine cross-linked dermis, polypropylene mesh, and polyester sutures in pelvic reconstructive surgery

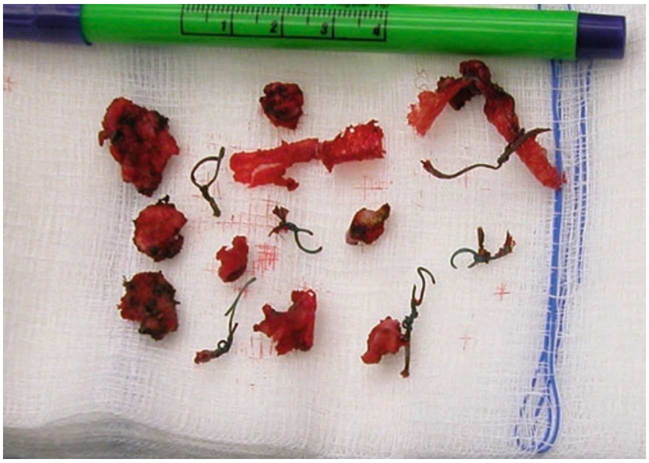

Fig. 20.10 Surgically explanted material from the case in Fig. 20.9

Conclusion

Various meshes and bolsters are used to add strength to pelvic organ prolapse repairs. Abdominal hernia literature has demonstrated the effectiveness of these materials in the ventrum of the body. Unfortunately, the vaginal vault hosts a varied and florid bacterial ecology. Bacterial colony counts in the vagina are higher than anywhere in the body with the exception of parts of the gastrointestinal tract. Not surprisingly, complications are relatively frequent in this setting.

Surgeons should use meshes and bolsters with all of these limitations in mind and should mange exposures, erosions, scar contractures, abscesses, pain syndromes and fistulae accordingly. Copious irrigation and meticulous biomechanically sound surgical technique at the time of surgery is essential to prevention. Acquaintance with electrical microdissection and basic plastic surgery techniques will help preserve and restore depth, axis, and caliber in the vaginal vault if problems do arise. If a surgeon implants a foreign material in the body for a nonlife threatening issue, she/he should have the ability to remove that same material if a significant complication occurs.

Removal of Vaginal Mesh After Cure of Genital Prolapse and Incontinence: A Case Series of 104 Operations

A retrospective continuous series study was conducted in the University Hospital of Caen, including all patients treated for operative removal of vaginal mesh between January 2004 and December 2008. Operations have been performed by the surgeon team of the gynecological department. Data were collected from medical records and are included in this report. All patients were evaluated for complications and outcome during their hospital stay and 6 weeks after the operation. Additional follow-up and assessment was done upon need and with symptoms.

The aims of the study were to evaluate the pre- and postoperative complications of the procedures, operation time, duration of hospital stay, and outcomes with regard to anatomical and functional results.

Surgical Techniques

Release of suburethral mesh can be accomplished under local anesthesia in most patients. A small sagittal cut is performed 1 cm under the urethral meatus in order to reach the sling that can be palpated as a band, followed by a sharp cut of the band in one of the band arms. The vagina is closed with two or three separate absorbable sutures.

Partial removal of the mesh is performed under general anesthesia in order to have a good exploration of the vicinity of the mesh. The extruded part of the mesh is removed and the remaining mesh is carefully examined for signs of infection. The vagina is closed with running locked absorbable suture.

Complete removal of vaginal mesh is performed under general anesthesia. For complete removal of the anterior mesh, a midline full thickness incision is performed on the anterior vagina, extending up to 2 or 3 cm from the urethral meatus. The bladder is dissected away from the vaginal wall and the paravesical fossae are opened until the ischial spine and the arcus ten. Data were collected from medical records and included tendineus of the levator ani are reached. The body of the mesh is dissected carefully and removed from under the bladder and the arms are pulled from the paravesical fossae. For complete removal of a posterior mesh, a midline full-thickness incision is performed on the posterior vagina extending up to 1 cm from the uterine cervix or hysterectomy scar. The pararectal fossae are opened until the ischial spine and the sacrospinous ligaments are reached. The body of the mesh is dissected carefully and removed and the arms are pulled from the pararectal fossae. In case of infection, an attempt to remove all the mesh, including both the body and arms, is made along with all the abnormal discharge or pus. The vagina is closed with running locked absorbable suture.

Laparoscopy to remove Tension-free Vaginal Tape (TVT) is made through extraperitoneal insufflation in order to reach the prevesical space of Retzius. The dissection is continued until Cooper's ligaments are reached and the TVT band is dissected and can be removed. Often, the most densely adhered portion of a TVT is at the level of the rectus abdominis muscles, where care must be taken to avoid bleeding. In cases of mesh adhesion, the remaining mesh can be removed through a vaginal approach as described in previous paragraphs.

All the removed meshes should be sent to histological and bacterial examination.

Results

Between January 2004 and December 2008, 83 patients had operative excision of vaginal mesh in our gynecological department. Seventeen patients (20.5%) needed more than one operation and overall, there were 104 operations for mesh removal. The mean age was 62 years (range, 34–84), mean operation time was 21 min (range, 5–65) and mean hospital stay was 3 days (range, 1–10). The different types of the primary operations for installation of the mesh, the type of the operative mesh removal, and location are presented in Table 20.3. The time interval between the installation and the removal of the mesh, as a function of the indications is described in Table 20.4. The indications for the interventions were in certain cases multifactorial. Erosion, without signs of infection, was the reason for 44 operations; 24 were vesico-vaginal mesh, 13 were recto-vaginal mesh, 7 were suburethral slings, and one was within the bladder. Infection was described in 30 cases, involving abnormal secretion, pus, and fistulization to the skin in some cases. Among the infection cases, only seven had positive culture. There was no detection of a specific pathogen (two *Escherichia coli*, one Colibacille, two *Staphylococcus aureus*, one Fusobacterium, and one *Streptococcus constellatus*). Five had abscess as a presenting symptom, three had fever, and one had an infected hematoma 3 weeks after the primary operation. Nine interventions for mesh removal were because of pelvic pain; among them, three described dyspareunia and one pudendal pain.

Perioperative complications occurred in two operations: one case of attempt to remove TVT by laparoscopy was converted to laparotomy because of difficult hemostasis. The other case was during resection of a retro-pubic intravaginal

Table 20.3 The primary mesh operation and mesh removal: type of operation, location, and indication

Primary mesh operation (*n*)	Type of operative mesh removal (*n*)	Location of mesh removal (*n*)	Indication (*n*)
Triple Operation for Prolapse with Prostheses (TOPP)[a] (31)	Partial removal (14)	Recto-vaginal (28)	Erosion (44)
Cystocele mesh (16)	Complete removal (61)	Vesico-vaginal (42)	Infection (30)
IVS posterior ± rectocele mesh (11)	Laparoscopy (5)	Suburethral (37)	Granuloma (10)
TVT/IVS retropubic (13)	Section (15)		Incomplete voiding (17)
TOT/TVT-O (21)	Undetectable mesh (2)		Pelvic pain (9)
Laproscopic Burch operation with mesh (1)	Uretrolysis (1)		Mal position (4)
Uretex (1)	Removal of Burch (1)		Suburethral collection (1)
Pelvicol (1)	Search for residual mesh (5)		Recurrent UTI (1)
Concomitant HVV (6)			

[a] TOPP operation include: cystocele, rectocele, and level 1 defect repair*IVS* intravaginal sling, *TVT* tension-free vaginal tape, *TOT* transobturator tape, *TVT-O* tension-free obturator tape, *UTI* urinary tract infections

Table 20.4 Distribution of the indications for mesh removal and the duration from primary operation

Indication / Duration	Erosion (44)	Infection (30)	Granuloma (10)	Pain (9)	Incomplete voiding (17)
Within 2 years	26	10	6	2	9
After 2 years	5	5	1	3	2
After 3 years	7	11	3	3	3
After 4 years	4	4	1	1	2
>4 years	2				1
Overall after 2 years (%)	18 (41)	20 (66.6)	4 (40)	7 (77.8)	8 (47)

sling (IVS) when a bladder injury was noticed and was sutured immediately. Postoperative complication occurred in 11 (10.6%) interventions: three had fever that resolved after antibiotic treatment. Five patients had postoperative hematomas; three went through reoperation for drainage of the hematoma, and two were managed conservatively. One patient required blood transfusion and one Venofer® (American Reagent, Shirley, NY) infusion. Among the postoperative hematomas, two occurred after complete removal of the mesh, one after laparoscopy, one after ureterolysis, and one after search for residual mesh. Other postoperative complications were: persistent voiding difficulties in one patient which required dilatation with Hegar's dilators under local anesthesia after 4 days. One patient had continuing bleeding and was reoperated for hemostasis. The patient with the bladder injury during section of retro-pubic IVS band had a postoperative complication with vesico-vaginal fistula and was reoperated after 2 weeks with no further consequences.

The four major types of operative mesh removal are presented in Table 20.5. Most patients had complete removal of the mesh. The mean operation time and hospital stay as a function of the indication for the removal of the mesh are described in Table 20.6. There were six postoperative complications in this group: two hematomas (one reoperated and one received blood transfusion), two postoperative fever, one voiding difficulty and one continuous bleeding. In the partial removal group, there were no pre- or postoperative complications. One preoperative complication occurred during laparoscopy and was difficult hemostasis which required laparotomy. Postoperative complication occurred in another patient and was hematoma in the Retzius space which was treated conservatively. Among the 14 patients that went through section of the band, one required a recurrence section of the trans obturator tape (TOT) in the contralateral side. Eleven sections were of suburethral slings and four were of the anterior arm of the cystocele mesh. Twelve out of 15 interventions (80%) were under local anesthesia. One pre- and postoperative complication occurred in this group: bladder injury which was sutured immediately. The patient developed postoperative vesico-vaginal fistula and was reoperated with no further complications.

Seventeen patients (20.5%) had more than one operation for mesh removal and there were 40 operations in this group. The indications for the primary and the sequential interventions are presented in Table 20.7. Twelve patients were reoperated twice, three went through three interventions, and two patients had four operations for the removal of all the mesh. Twenty six operations were at the same location, ten in different locations, and two patients were operated twice for excision of different primary meshes.

Recurrence of pelvic organ prolapse (POP) or stress urinary incontinence (SUI) was observed in 22 patients. All prolapse recurrences were of cystocele. Overall, there were 42 operations for removal of vesico-vaginal mesh and eight cases (19%) of cystocele recurrence. Six recurred after complete removal and two after partial removal. Seven patients were reoperated: three with reimplantation of vaginal mesh and four received laparoscopic sacrocolpopexy. SUI was recurred in 14 patients (37.8% of all suburethral sling interventions): eight after complete removal of suburethral sling, four after section of the band, one after laparoscopy excision of TVT, and one after partial removal. Ten patients were reoperated: four with TOT and six with retro-pubic IVS sling.

Discussion

The high recurrence rate of POP after repair with autologous tissue, along with the introduction of mesh to treat incontinence by the TVT[13] resulted in dramatic progress and development of vaginal mesh surgeries. Since the commercial kits for vaginal mesh surgeries are very popular today, there are many untrained surgeons placing vaginal mesh to cure prolapse and SUI, lacking the right anatomical knowledge of the pelvic floor. This situation enhances the possibility for complications, sometimes very severe. Furthermore, when complications do occur, this lack of knowledge and experience can contribute to significant morbidity if it is not treated in the right way. Our aim was to reveal the way we treat vaginal mesh complications in a trained tertiary referral center.

Table 20.5 Characteristics of the major types of operative mesh removal

Intervention	Patients (*n*)	Operations (*n*)	Mean age (year)	Mean op time (min)	Mean hos stay (d)	Indication (*n*)	Complications		Recurrence (*n*)
							Pre-op	Postop	
Complete removal	57	61	62.8	21.08	3.1	Erosion (31) Infection (22) Mal position (3) Granuloma (5) Incomplete voiding (2) Suburethral collection (1)	Non	6	Cystocele (7) SUI (8)
Partial removal	14	14	60.9	14.5	2.5	Erosion (11) Granuloma (2) Dyspareunia (1)	Non	Non	Cystocele (1) SUI (1)
Laparoscopy	5	5	57.4	46.25	5.2	Pelvic pain (4) Rec. UTI(1)	1	1	SUI (1)
Section	14	15	61.8	9.2	1.3	Incomplete voiding (13) Pain (2)	1	1	SUI (4)

op operation, *hos* hospital, *min* minutes, *d* days
UTI urinary tract infection, *SUI* stress urinary incontinence

Table 20.6 Complete removal group: mean operation time and hospital stay as a function of the various indications.

Indication (*n*)	Mean op time (min)	Mean hos stay (days)
Overall (61)	21.08	3.1
Infection (22)	27.5	3.7
Erosion (31)	18.3	2.7
Mal position (3)	9.3	2

op operation, *hos* hospital, *min* minutes

Table 20.7 Recurrent cases of operative mesh removal: indications for the primary and the sequential interventions

Primary operation (*n*)	Secondary operation (*n*)
Erosion (10)	Recurrent erosion (5) Infection (5)
Infection (4)	Recurrent infections (4)
Granuloma (1)	Infection (1)
Incomplete voiding (1)	Continuous incomplete voiding (1)
Recurrent UTI (1)	De novo pain (1)

UTI urinary tract infections

Over 4 years, we had 104 operations for mesh removal. Not all mesh complications required complete mesh excision. Fourteen patients had partial removal of the mesh: 11 (78.5%) had a small erosion, two had granuloma, and one dyspareunia. For ten patients (71.4%), removal of the extruded part was sufficient, as known from other publications.[14] Four patients with simple erosion, which went through partial removal, needed reintervention: three because of infection and one with recurrence of erosion. All the secondary operative mesh removals in this group were complete mesh excisions. Altogether, 17 patients (20.5%) went through more than one operation for mesh removal and five were involved in more than two operations. These five patients had infection which recurred a few times until a complete ablation of the mesh was achieved. An unknown phenomenon was observed in two patients; although there were signs of repeat infection, no mesh was detected in reexploration. In the first patient, the primary mesh removal was of the vesico-vaginal mesh, and the repeat signs of infection were in the recto-vaginal mesh, and none was detected in the operating room. The second patient had a partial removal of mesh because of granuloma and again, the mesh was not detected when signs of infection recurred. In both cases, the mesh was polypropylene, which is nonabsorbable. In both cases, the interventions were longer than the average operation time (30 and 50 min) and the vagina was cleaned from pus with no further recurrence.

The most frequent mesh complications described in the literature are; erosion, infection, pain, and shrinkage of the mesh.[14-16] The distributions of mesh complications, which were the indication for mesh removal in our series, were as expected. The most frequent complications were erosion and infection (42% and 28.8%, respectively). Incomplete voiding was the indication in 44% of suburethral slings operation and was the leading cause for section of the band. Pain was the reason for nine interventions, although only in six it was the major cause. In four patients, pain appeared after placement of TVT and was the cause for 80% of laparoscopy removal of TVT. Mesh folding was not a cause in our series, although described in other series for mesh excision.[17] Four cases of mal-position were noticed and were the reason for recurrent prolapse and recurrent UTI as an indication for mesh removal, but there was no mesh folding or shrinkage. Shrinkage of the mesh is a complication described widely[14,18] and can result in severe deformation of the vagina causing dyspareunia, defecatory and urinary dysfunction. Since in our department there is a separation between anterior and posterior compartments during prolapse repair, it seems to reduce the risk for shrinkages of the mesh. Another severe and serious complication described in other series is the formation of fistula between the vagina and the rectum or bladder.[17,19] We had 104 operations for mesh excision, none were because of fistula. It appears that in a trained center, such a complication after mesh installation is very rare.

Most of the publications today, which report outcomes of vaginal mesh operations, are of short and medium-term follow-up. Thus, most of mesh complications appear to occur within the first year after the operation. In our series, we found mesh complication even 8 years after installation of the mesh. Moreover, 57 of the operative mesh removal (54.8%) were performed more than 2 years after the primary operation. Eighteen out of 44 patients (41%) with erosion were detected after 2 years, 20 out of 30 (66.6%) with infections, 4 out of 10 (40%) with granuloma, 7 out of 9 (77.8%) with pain, and 8 out of 17 (47%) with incomplete voiding. The most surprising finding was the high percentage of infections which were detected more than 2 years after the primary operation. In most cases of infected mesh, the cultures were sterile and only in seven cases there was detection of a pathogen. The diagnosis of infection was made upon abnormal secretion, pus, and fistulization to the skin in certain cases. In the literature, there is no detection of a special pathogen[20] and most infections are described within the first year. The causes for graft infection have been studied since the beginning of graft use[21] along with the debate regarding its nature. It seems, according to the current knowledge and our findings, that the infections are chronic in nature, without a specific pathogen and in most cases sterile by culture. More biological research is needed in order to try and detect responsible pathogens. On the other hand, the lack of a pathogen in most cultures may imply that these patients have chronic inflammation and foreign body reaction to the mesh which may play a role in the development of these complications and can explain the late presentation after the primary operation.

The operation time was 21 min on average, quicker than what is published in other series.[19] We assume that this fact is related to our large experience with mesh operations and the right knowledge to treat complications. It was interesting to observe that the mean operation time was different according to the indication. When the indication was malposition, mean operation time was 9.3 min, 18.3 min when the indication was erosion, and 27.5 min for infected cases. The mean hospital stay was found to be a function of the indication, accordingly, with longer hospitalization for infection cases.

Recurrence of POP or SUI after ablation of the mesh is an interesting issue. Out of 70 operations for vaginal mesh removal (28 recto-vaginal and 42 vesico-vaginal), eight patients (11.4%) had recurrence of prolapse, and all were cystocele. There was not a single case of recurrence of posterior or central compartment. With regard to the cystocele recurrence, 8 out of 42 operations for removal of vesico-vaginal mesh (19%) had recurred cystocele. This observation is consistent with the known fact that most recurrences are in the anterior vaginal wall.[22] Nevertheless, in 80% of these operations, there was no recurrence although the mesh was removed. Recurrence of SUI after removal or section of suburethral sling was even more pronounced. There were 14 cases (37.8%) of SUI recurrences after 37 suburethral sling operations. Four were after section of the band, nine after complete removal, and one after partial removal. Thus, recurrence of SUI is much more frequent than recurrence of POP after mesh removal. In all recurrences, POP and SUI, no correlation was found between mesh removal within the first year and recurrence. It seems to us that the body's reaction to the mesh after prolapse operations is sufficient enough to hold and prevent prolapse recurrence in most cases, even after the removal of the mesh. It might be that the narrow suburethral sling is not large enough to provoke a sufficient body reaction to last after the removal of the mesh, and once the band is removed, the SUI is likely to recur.

Conclusion

In a trained center, mesh removal is a quick and safe procedure with very few pre- and postoperative complications. Mesh complications may occur frequently more than 2 years after the primary operation, exceeding the current period known from short follow-up publications. Recurrence is mostly associated with SUI and less with POP. We encourage surgeons to further expand their anatomical knowledge, obtain specialized training for each mesh placement technique, and be aware of known risks, as recommended in the Food and Drug Administration public health notification of October 2008.[23] The result will be reduction of serious complications after mesh surgeries and an increase in the knowledge and expertise of how to treat complications when they do occur.

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Recurrence in Prosthetic Surgery

21

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and Bernard Jacquetin

Introduction

As we shall be seeing through a review of the literature, recurrence following prolapse surgery is, at least as regards certain indications and techniques, reduced by the placement of what, in this chapter, we will be calling an implant. Recurrence does, however, happen and we will look here at its incidence, particularities, and management. There are several benefits in investigating the topic. Firstly, recurrence after implant placement occurs in different forms, both anatomically and with regard to its development. Secondly, the diagnosis is often misleading and we will be examining the advantages of ultrasound in this situation. Finally, while an implant is often used when there is a recurrence of prolapse, what treatments can be considered in the event of postimplant recurrence? This is a fresh issue that is rapidly becoming more important and we will attempt to provide an answer both through data in the literature and long clinical experience in the use of implants.

Current Situation

Definitions

Defining recurrence might seem superfluous, but perusal of articles shows that the word is open to many kinds of definition. It is, however, crucial to give a precise meaning for recurrence, in order to interpret the raw results of the studies we read and also to have a precise idea of the efficacy of treatments and thereby to give proper advice to our patients. How should we consider treatment for cystocele that claims a 90% success rate, but which, upon analysis, results in a rectocele or a hysterocele in 40% of the cases? What functional outcome and patient satisfaction can be expected?

"Recurrence," "development," "recurrence in another compartment," "de novo prolapse," "decompensation," "successful," "failure," "cure," "improvement," are all terms and situations that are sometimes hazy and which need to be defined. The lack of homogeneity in the literature makes the precise incidence of such situations also difficult to determine. It is up to us to present results in a more uniform and more transparent way.

We shall first look at the definitions drawn up by Weber et al.[1] for America's National Institutes of Health to agree on standard terms for defining conditions and outcomes, although these definitions are unfortunately not widely used. The authors' definitions, based on the ICS anatomic classification,[2] are as follows:

- Optimal anatomic outcome (cure): stage 0
- Satisfactory anatomic outcome (improvement): stage I prolapse
- Unsatisfactory anatomic outcome (persistence or recurrence, failure): stage II or higher or no change or aggravation compared to the preoperative stage

It should be emphasized that this terminology does not factor in the compartment treated. It could therefore be considered that a postoperatory stage II is a failure, irrespective of the compartment treated. The unsatisfactory outcome group will therefore include a number of different clinical situations, which it is interesting to differentiate. An unsatisfactory outcome might concern the compartment treated and could be described as recurrence or persistence, depending on when the reaction occurred. The unsatisfactory outcome might concern an untreated compartment and we will call this decompensation. It could involve the development of a pre-existing prolapse where no surgery has been performed or the appearance of a prolapse in an untreated compartment, in other words a de-novo prolapse (Fig. 21.1).

For the sake of clarity, we will from here on use the words failure, recurrence, or decompensation, depending on the case. However, leaving aside the definitions, proper data can be obtained only through transparency and completeness of

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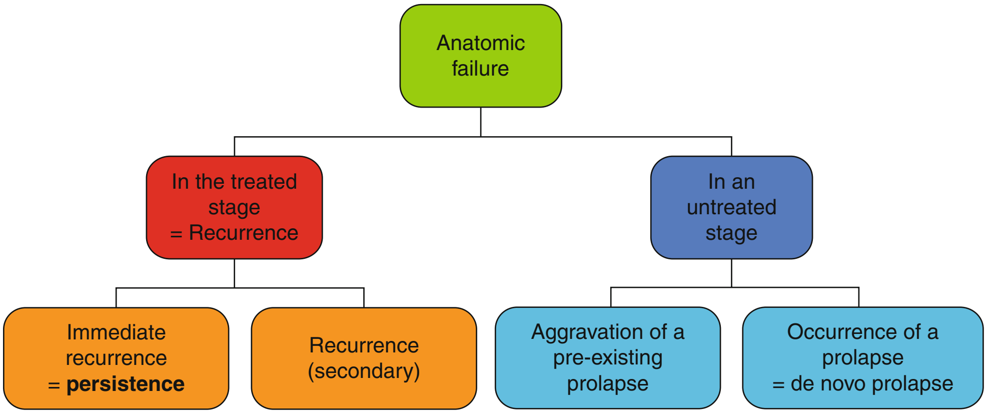

Anatomic failure
In the treated stage = Recurrence
In an untreated stage
Immediate recurrence = **persistence**
Recurrence (secondary)
Aggravation of a pre-existing prolapse
Occurrence of a prolapse = de novo prolapse

Fig. 21.1 Terminology

results. It is important when assessing implants to know the proportion of each of these situations in the unsatisfactory outcomes. A good number of articles claiming to follow the guidelines set out by Weber et al. only detail the recurrence for the compartment treated and do not give any information as to the incidence of the other causes of failure. Nevertheless, the knowledge of the proportion of decompensations resulting from an imbalance caused by the placement of an implant could provide useful information for developing the concepts of selective or complete repair of the pelvic floor.

To give an idea of the lack of uniformity in the definition of recurrence, despite the efforts that have been made as regards discipline and standardization, let us examine some of the variations used in a number of randomized studies since Weber et al.'s paper.

Nguyen et al.,[3] Paraiso et al.,[4] Meschia et al.[5] considered the outcome for the treated compartment only and included in the same group of "cured" patients those with an optimal outcome and those with a satisfactory outcome, according to Weber et al.'s classification.

Sivaslioglu et al.[6] considered that the surgical outcome is "acceptable" if the treated compartment is at stage 0 or I. For Hiltunen et al.,[7] the outcome is considered for the treated compartment, while for De Tayrac et al.,[8] a successful anatomic outcome (secondary endpoint) was not defined.

Functional Aspect

We have given above the definition of anatomical recurrence. A further aspect of recurrence, on the periphery of the subject but still essential, is the symptomatology that might be associated with it. Experience shows that many anatomical outcomes that are disappointing from the surgeon's point of view are associated with patient satisfaction as there are no symptoms.

Data in the literature are patchy, especially as regards the correlation between the anatomical outcome and the functional symptoms or recurrence of prolapse. Although the issue is far from being settled, we will give a couple of examples to throw light on the situation.

Prolapse Recurrence and Symptoms

Several studies[9-13] show, for nonoperated populations, a correlation between the prolapse stage and certain symptoms. To simplify, despite imperfect sensitivity and specificity, it would appear that the best correlation is for bulge-type symptoms with a prolapse that reaches or extends past the hymen, a fairly intuitive approach.

The symptoms relating to recurrence are less obvious and may differ according to the associated anatomic compartment. The findings of randomized studies concerning cystocele treatment[3,5-7,14] show that although there was a significant improvement in postoperative functional scores, there was virtually no significant difference in the groups with and without implant, although there were significant differences in the groups' anatomic outcomes. These results might be due to either the studies' lack of power, as they were designed to reveal an anatomic difference, or the paucisymptomatic nature of the anterior recurrences, as noted by Weber et al. in another prospective study with high rates of recurrence. In the only study where the information is available,[7] it can be seen that only a minority of the recurrences (the time to occurrence of which is not known) requires reoperation within the first 12 months. Only one of the seven recurrences within the group with implant and 1 out of the 37 recurrences in the group without implant required reoperation within the first 12 months, a revision rate of 4.5% for the recurrences. Rectocele recurrences appear, on the other hand, to be more symptomatic. In the randomized study conducted by Paraiso et al.[4] on three surgical techniques for rectocele repair, the functional symptoms were analyzed at 12 months. There was no significant difference between the three treatment groups and the functional outcomes assessed using PFDI 20 were combined, allowing analysis of 106 patients.[15] The analysis showed a clear reduction in the risk of bothersome terminal constipation (OR =.017, 95% CI: 0.03–0.9) and bothersome incomplete emptying (OR = 0.1, 95% CI: 0.01–0.52) in cases of satisfactory or optimal anatomic outcome. The results were not included in the meta-analysis performed by Maher et al.,[16] which drew no conclusions as to the effect of surgery on bowel functional symptoms.

Recurrence and Sexual Outcomes

With regard to the link between sexual outcome and recurrence, Altman et al.[17] did not find any correlation at 1 year between an optimal or a satisfactory anatomical outcome and the PISQ-12 score, in a prospective multicentre series of 84 sexually active patients treated by Prolift®, an outcome in accordance with other publications after nonprosthetic surgery.[18] Although the link with the anatomic outcome has not been established, several studies have found unfavorable scores in the case of prolapse[19] and an improvement in postoperative sexuality.[20,21] Conversely, for some patients, postoperative sexuality appears to remain unchanged[22-24] or altered.[17,25] However, the question of sexuality after prosthetic repair is most likely to result from potentially associated reactions such as dyspareunia and prosthetic retraction[26,27] rather than the recurrence itself, but these issues are not addressed in this chapter.

If on top of these conflicting outcomes, we add the observation made by Wren et al.,[28] according to which an optimistic state of mind on the part of the patient significantly reduces prolapse symptom severity or the finding that the change in postoperative sexual scores reported by Altam et al.[17] in fact concerns the relational and emotional issues with the partner, or again the importance of underlying relational and sexual disorders reported by Gauruder-Burmester et al.,[24] then it can be considered that it is essential to remain highly meticulous but cautious in evaluating the functional aspects of prolapse and recurrence.

Incidence

There is as much variation in the exact incidence of recurrence as in its definitions. In addition, the data in articles are too often limited to the treated compartment, which is insufficient for evaluating the overall management of our patients and their perineum. Nevertheless, despite these uncertainties and leaving aside arguments over numbers, one fact seems to stand out: the placement of an implant reduces recurrence. We see that there are an increasing number of arguments to support this conclusion and we will attempt to summarize them.

First, the low recurrence rate of sacrocolpopexy constitutes, without a doubt, the strongest historical argument. The quasi systematic failure of suture sacrocolpopexy, resolved by implant placement, the similarity in the outcomes of sacrocolpopexy by laparotomy and by coelioscopy despite the very different techniques, the equally encouraging outcomes achieved with alternative techniques using the abdominal route, without sacrocolpopexy but with a mesh reinforcement,[29] offer indirect arguments to support the hypothesis through which the efficacy of sacrocolpopexy is related more to the implant than to the technique. Without delving into the details of numerous publications, the recurrence rate after sacrocolpopexy can be estimated between 0% and 22% for the middle compartment and between 0% and 42% for recurrence for any compartment.[30,31]

Moreover, in anterior prolapse repair, the results of trials randomizing surgery with implant versus surgery without implant, consistently (despite the different techniques and materials) support a reduction in recurrences in techniques using an implant (Table 21.1 and Fig. 21.2).[33]

A meta-analysis by Jia et al.,[34] used a number of statistical methods to combine published results. Thirty studies on cystocele repair were identified, involving 2,472 women with a mean follow-up period of 14 months. The combined objective efficacy produced the following crude failure rates:

- 28.8% without mesh
- 23.1% with absorbable biological mesh
- 17.9% with absorbable synthetic mesh
- 8.8% with nonabsorbable synthetic mesh

Meta-analysis assessment and indirect comparison suggest an increased failure rate with absorbable biological or synthetic mesh compared to nonabsorbable mesh (Respectively, OR: 4.12 [IC: 2.2–7.7] and OR: 2.97 [IC: 1.83–4.6]). The authors also found a lower relative risk of recurrence in cases where mesh was used (all types): RR = 0.48 (IC: 0.3–0.72).

However, these data supporting prosthetic repair in anterior prolapse do not take into consideration decompensation, which is a significant factor in exploring options between unicompartmental and complete repair. The decompensation rate is seldom available. It is, therefore, reported only in small populations in the systematic review conducted by Jia et al.[34] and is at 13.8–17.8% in anterior mesh repairs and unavailable in other types of repairs.

The failure rate for the middle compartment (not including sacrocolpopexy, for which the outcomes are given above) is estimated between 0.9%[35] and 26%[36] for posterior IVS. Highly different techniques featuring more or less systematic colporrhaphies, or prosthetic repairs in other compartments[37,38] make it difficult to distinguish between a recurrence and decompensation.

Implant placement in the posterior compartment (using a variety of materials and techniques) is the reason for

Table 21.1 Recurrences and decompensations, randomized studies with or without anterior mesh

Reference ("population" at "mean follow-up")	Recurrence: cystocele with implant versus without	Posterior decompensation: rectocele	Apical decompensation	Reoperation for recurrence or decompensation	Failure: recurrence or decompensation
Nguyen and Burchette (76 at 1 year)[3]	13% versus 45% $p = 0.005$ (OR: 5.3 [1.17–17]) Stage ≥ II (POP-Q)	/	0 versus 0	0 versus 121	/
Sivaslioglu et al. (85 at 1 year)[6]	9% versus 28% ($p < 0.005$)	/	/	/	/
Meschia et al. (201 at 15 months)[5]	7% versus 19% $p = 0.019$ (OR: 3.13 [1.26–7.78]) Stage ≥ II (POP-Q)	3 versus 8 (NS)	3 versus 3 (NS)	/	/
Hiltunen et al. (201 at 12 months)[7]	6.7% versus 38.5% $p < 0.001$, Stage II or III (POP-Q)	/	Mean C value:–7.5 versus –7.2 (NS)	1 versus 1	/
Weber 2001 (83 at 23 months)[14]	58% versus 54% (NS) Stage ≥ II (POP-Q)	/	/	/	/
Sand 2001 (143 at 1 year)[32]	25% versus 43% $p = 25\%$ Stage II Baden-Walker	8.2% versus 10% $p = 0.71$	/	/	/

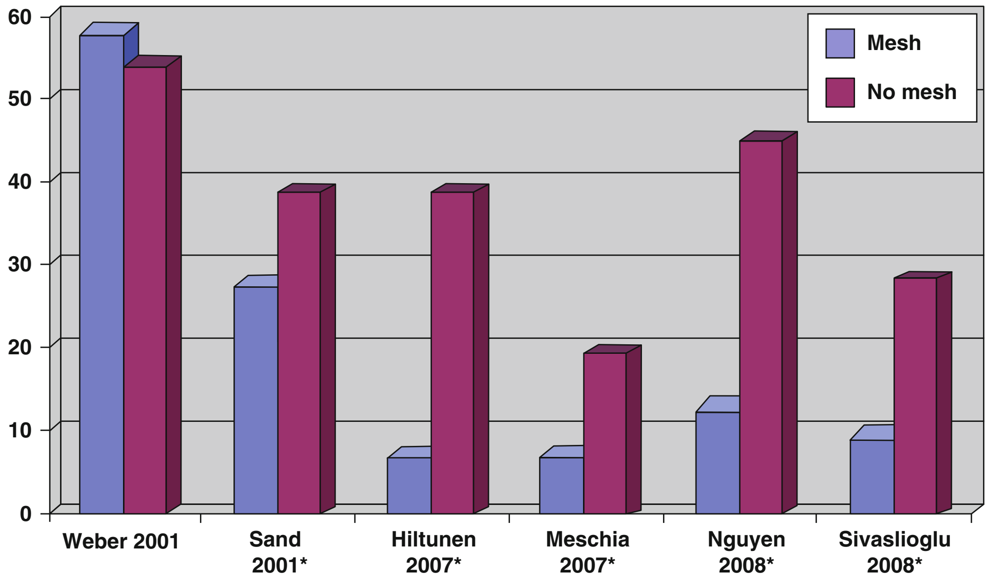


Fig. 21.2 Recurrences, randomized studies on the anterior compartment. *Significant difference

recurrence in 8.2%[32] to 46%[4] of cases. Once again, the combination with other procedures makes it impossible to determine the rate of decompensation. Conflicting or insufficient results from studies included in two other previously mentioned meta-analyses[16,34] mean that it was not possible to draw a conclusion as to the benefit of implant placement in the posterior compartment.

Our experience has shown that in a follow-up period of at least 1 year, in a series of 107 prosthetic repairs by vaginal route,[39] the recurrence rate on the treated compartment is 15.9% (17/107). Decompensation occurred in 17.6% (9/51) of unicompartmental prosthetic repair cases; in this study one third of failures were due to decompensation. It is interesting to note that decompensation occurs much more often in the form of anterior recurrence after isolated posterior repair than the other way round. We observed decompensation in 38.9% (7/18) of the cases after posterior implant placement and 6% (3/33) following anterior implant placement.

Risk Factors

The general risk factors for prolapse recurrence, considered controversial by some authorities, are not addressed here.

We only cite low age, the severity of the prolapse, obesity, past history of prolapse repair or incontinence, cough or chronic constipation, and conjunctive tissue anomalies. Their link to recurrence in the specific case of prosthetic repair is only probable.

On the other hand, a number of factors for postimplant recurrence need to be considered.

Retraction seems to be an independent risk factor for recurrence in certain studies that have investigated it.[40,41] One of the difficulties in assessing the importance of this factor is the lack of standardized assessment criteria and an objective measuring instrument. Certain teams, including our own, are working in this direction and a classification system is due to be published soon. We discuss below how this factor influences recurrence and how it can be assessed by ultrasound.

The surgeon's expertise is a potential risk factor for recurrence. It is clear that rigorous training in prolapse surgery in general and especially in the technique utilized is an indispensable prerequisite. Nevertheless, once this hurdle has been overcome, it is interesting to note that in our experience, the utilization of implants by vaginal route with an introduction kit does not yield a significant difference in terms of recurrence (or other complications) between the population who underwent surgery performed by junior surgeons and those who underwent surgery performed by senior surgeons.[42]

Finally, the role implant exposure plays in recurrences needs to be monitored on a long-term basis. Undeniably, the treatment of implant exposure by excision, sometimes repeated, could create significant defects in implant coverage and could even lead to complete removal of the implant. These situations therefore constitute a risk of recurrence, even if their importance remains to be assessed through patient follow-up. Inasmuch as exposure is, in certain studies[43] related to the surgeon's experience, it can be seen how much this can indirectly affect recurrence.

This brief overview of the current situation regarding recurrence does not reveal certain evolutive and anatomical features in recurrence after implant placement. These features are, however, interesting from a physiopathological perspective, and have clinical and therapeutic significance.

Features of Recurrence After Implant Repair

Evolutive Features

Although recurrence is possible when there is no implant, certain cases of recurrence after mesh repair occur extremely early, suggesting a technical failure.

It seems that the incidence of recurrences stabilizes in the medium-term after the first year of follow-up (unpublished follow-up data at 3 years after an implant using the TVM technique, which are consistent with the results observed by Weber et al. [14] and Paraiso et al.[4]).

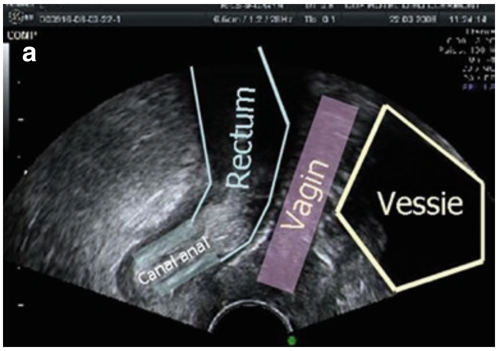
As we mentioned earlier, recurrence after implant placement is probably influenced by two parameters – retraction and exposure. Both are dynamic reactions which often occur early but sometimes not for several years. This little-known aspect of secondary recurrence still needs to be evaluated through long-term follow-up of our patients.

Ultrasound Aspects of Prolapse and Implants

Over and above the conventional clinical distinction between medial and lateral cystocele, ultrasound makes it easier to understand prolapse[44,45] and recurrence after implant placement.

Once the anatomical landmarks are detected for a UroGynaecology ultrasound test,[46] as illustrated in Fig. 21.3, it is easy to conduct a postoperative identification of synthetic implants, which is what we have been doing since 2000 and as recommended by other authors.[47,48] Polypropylene is visualized as a hyperechoic edge under the vaginal surface at its visceral interface. In tangential incidence, the net structure of the mesh is also visible, confirming, if necessary, the presence of the implant.

Ultrasound facilitates localization of the prosthesis in relation to the vagina and adjacent organs, analysis of the appearance of the implant itself (distribution and thickness), and the formulation of a physiopathological hypothesis for



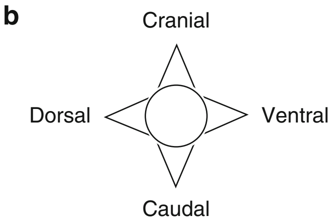


Fig. 21.3 Echo-anatomical landmarks (anal canal, rectum, vagina, bladder)

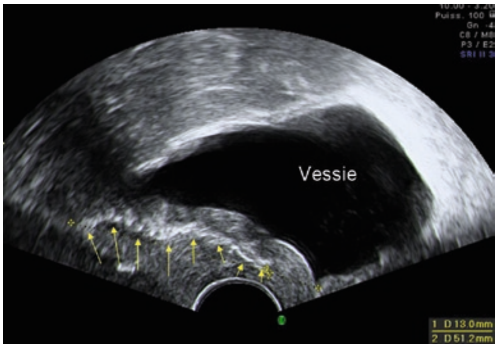


Fig. 21.4 Sagittal section of an intervesicovaginal implant by 2D ultrasound by vaginal route. Note the distribution of the implant (*arrows*) from the urethrovesical junction to the subperitoneal space (posthysterectomy); measurement 1 (13 mm) represents the distance separating the lower edge of the implant from the urethrovesical junction. Measurement 2 (51 mm) represents the length of the implant. Anterior section-Bladder-Posterior section

each recurrence that, as we shall see, will provide a guide for the treatment (Figs. 21.4 and 21.5).

A detailed standard ultrasound test is possible, whether implant placement was transvaginal or transabdominal.[47-50] A three-dimensional test is also possible although we consider it is more difficult to perform and interpret.[46,50]

Physiopathological Approach and Anatomical Types of Recurrence

The clinical study and ultrasound imaging of prosthetic repairs can reveal the various recurrence mechanisms, shown by different anatomical features (Table 21.2).

Firstly, we can identify recurrences by “a defect in implant coverage.” This refers to site-specific recurrences in a compartment, which in theory was treated, but where a recurrence took place where there was no implant coverage. This recurrence through lack of coverage can be explained in several ways:

- The implant is well anchored but too small and does not cover the full anatomic defect. The recurrence occurs along an edge of the implant.
- The implant retracted, allowing a hernia in the noncovered area. In this case, it could involve atypical location recurrence, which could, for example, be very lateral in the case of transversal retraction (“string effect”) producing a paravaginal defect. Recurrence can also occur caudally or cranially (more rarely) with regard to a craniocaudal retraction. Retraction is common in implants, and can exceed 50% of the implant length.[47]

In a study conducted on 91 patients who underwent surgery for anterior and/or posterior mesh reinforcement with the Prolift® kit, we compared, at 12 months postoperative or more, the findings of the clinical examination with ultrasound imaging of the implants. We noted that the thickness of the implant on the ultrasound measured in a sagittal section was significantly correlated with the percentage of implant retraction, as estimated by vaginal palpation.[41] Furthermore, the extent of implant retraction was significantly correlated to recurrence. The lack of implant coverage in the distal section of the anterior and posterior vaginal wall was correlated with a recurrence. In most cases, the recurrence was located under the lower edge of the prosthesis which retracted cranially, forming a “low” cystocele or a “low” rectocele. These findings support the physiopathological hypothesis,

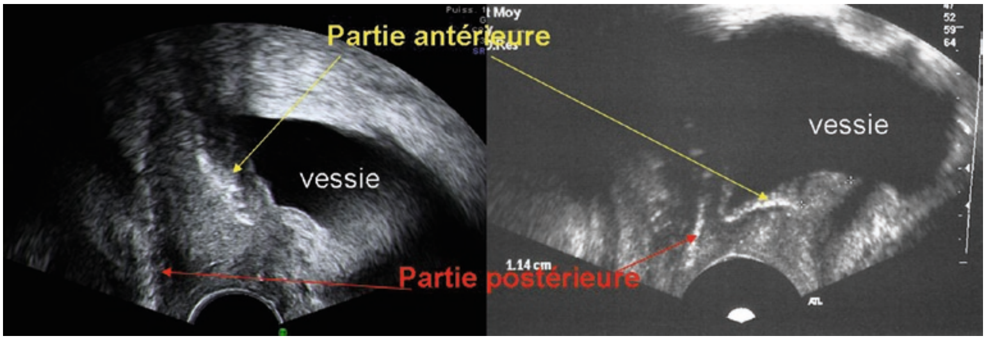


Fig. 21.5 Ultrasound image in a sagittal section of complete anterior (intervesicovaginal) and posterior (interrectovaginal) mesh reinforcement

Table 21.2 Physiopathological approach of recurrence

Recurrence	Anterior compartment	Middle compartment	Posterior compartment
Coverage defect	“String effect” Lateral cystocele Vaginal cystocele and trigonocele	Enterocele in inversion	“String effect” Lateral rectocele Vaginal rectocele
Fixation defect	Covered cystocele	“Piston-like effect” by the uterus	Covered rectocele
Decompensation	Cystocele Anterior enterocele	Cervix elongation	Rectocele

whereby retraction is one of the key mechanisms of recurrence (Figs. 21.6–21.8).

- One particular type of recurrence due to lack of coverage deserves to be discussed. This involves a low cystocele which forms under the caudal edge of the implant. This recurrence can be voluminous or it may remain limited in size, forming a "trigonocele." A trigonocele is seldom voluminous and can affect the anatomic outcomes of certain studies due to its low location and is often responsible for a Ba point at −1, related to the persistence of a significant cervico-urethral mobility. We consider that Sand et al.'s findings[32] whereby the suburethral tapes play a protective role in recurrent cystocele illustrates this mechanism. The phenomenon most likely causes a genuine recurrent cystocele to be confounded with cervicourethral recurrence and, without a doubt, has an adverse effect on certain anatomic outcomes. Following the results of a randomized trial,[14] Weber et al., who were actually behind the standardization of terminology for prolapse evaluation and recurrence,[1] noted with a certain indulgence that the strict definitions for anterior recurrence may be "mediocre criteria" for successful therapeutic outcomes. It is only through studying the development over time and the functional impact of these trigonoceles that we shall be able to determine the actual significance and seriousness of these recurrences (Fig. 21.9).

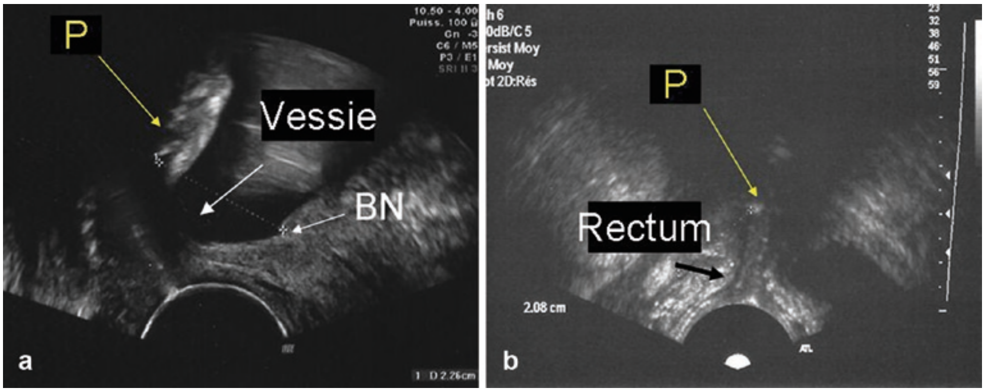


Fig. 21.6 Example of an anterior (**a**) and posterior (**b**) anatomic failure due to lack of implant coverage in the caudal section of the vagina

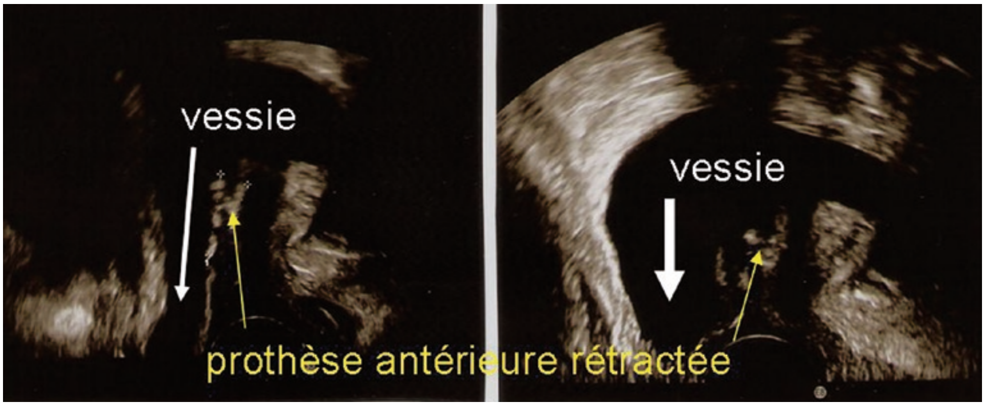


Fig. 21.7 Examples of recurrent cystocele above the anterior implant which retracted under the bladder neck. This situation is rarer in our experience

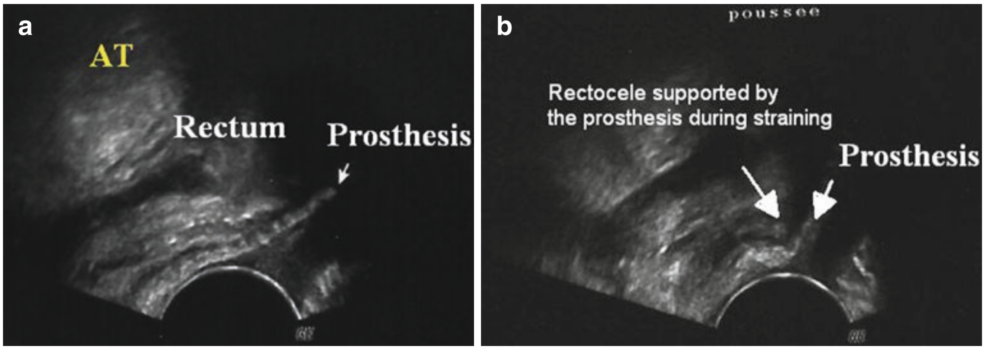
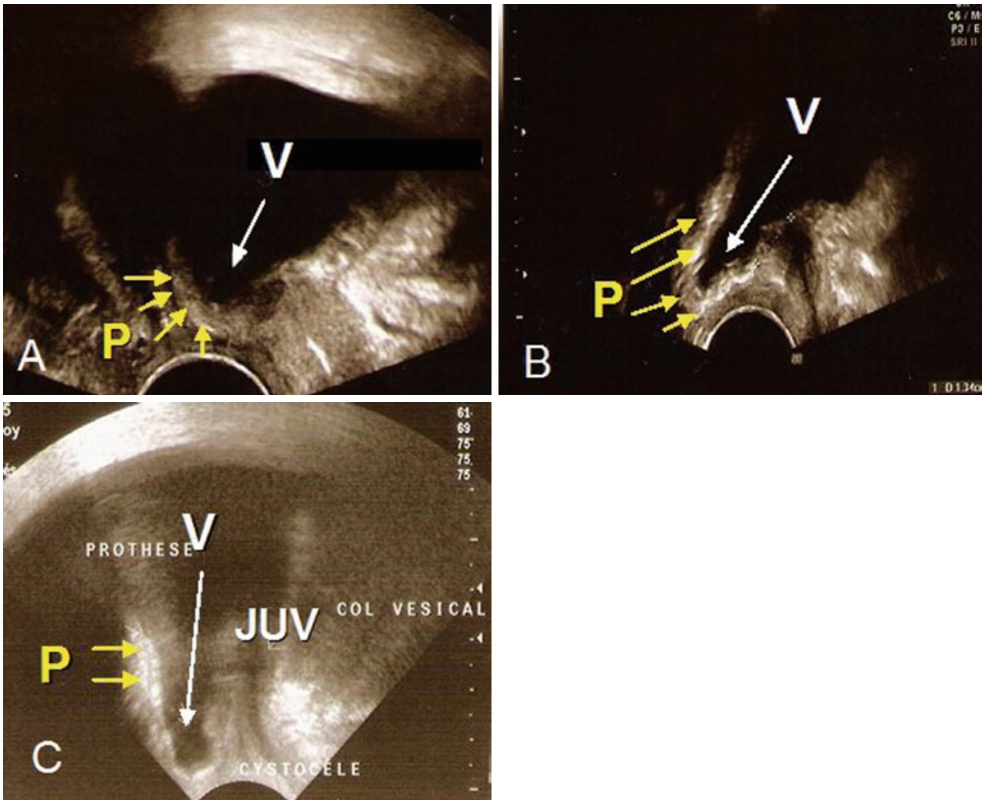


Fig. 21.8 Example of satisfactory support (Bp = −2) in the posterior stage after isolated posterior mesh reinforcement

Fig. 21.9 Different types of bladder supports (V) after intervesicovaginal mesh reinforcement (P). (**a**) Ba = −3, support during straining is effective. (**b**) Ba = −2, bladder support remains adequate in spite of a small tension defect in the mesh. (**c**) Ba = 0, there is a recurrent cystocele between the implant and the urethrovesical junction (*UVJ*) (trigonocele)



[redacted]

- Another anatomic feature also resulting from a lack of coverage is the occurrence of a prolapse, most often an enterocele, between separated posterior and anterior implants. In this case it is an enterocele through invagination.

It is also possible to identify a number of situations where recurrence is due to "fixation defects." Examples include:

- A "postage-stamp" implant that is too small and not anchored, which will shift with the organs and the vagina in the prolapse.
- Shek et al.[50] conducted a postoperative 3D–4D translabial ultrasound study on 46 patients who underwent surgery for a cystocele by transobturator anterior implant with 4 arms. These authors observed, after a mean follow-up period of 10 months, a recurrence in six patients (13%), including, in five cases, a cranial recurrence in relation to the implant with a change in the implant axis during the Valsalva maneuver. According to the authors, this would point to a detachment of the lateral prespinous arms of the implant. In our opinion, the hypothesis of an implant detachment does not appear to be very plausible. Tissue integration of the prostheses is such that a rupture does not seem likely. A possible explanation could, however, be a failure to secure the implant tightly enough in its cranial section, as we shall discuss below.
- A prosthesis that is of sufficient size and which covers the organs and the vagina can shift with them under mechanical stress. This can be due to an unanchored implant without lateral attachment or a prosthesis with attachment but which is not secured tightly enough. When there is a fixation defect related to an implant which covers the prolapse but shifts with it, we observed that the recurrences tend to remain "moderate" (Stage 2 POP-Q but intravaginal) and stable.[51] (Fig. 21.10).

The observation of this type of recurrence has led certain teams, including ours, to design implants of increasing sizes

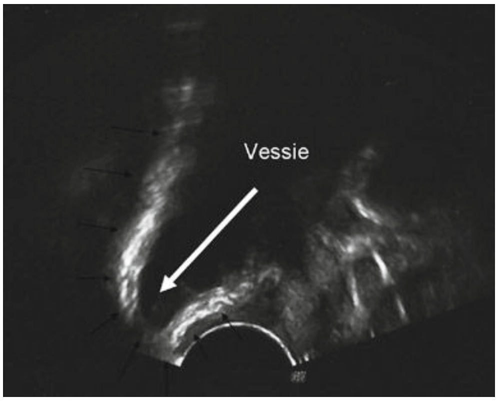


Fig. 21.10 Example of an anterior relapse following placement of an unanchored intervesicovaginal implant. Note that the bladder is well covered by the mesh (*arrows*), but it moves with the bladder during straining

which are anchored by various methods (Prolift®, Perigee®, Apogee®, Pinnacle®).

- An illustrative case of a fixation defect is the isolated recurrence of uterine prolapse. Fixation of the uterus to the mesh is usually performed by passing a suture in the cervix, the isthmus, or the uterosacral ligament and the implant. The anchoring can give way and produce an isolated recurrence of a "piston-like" effect by the uterus. The effect is accentuated by proper anterior and posterior correction by an implant. When the fixation is immediately defective, this type of recurrence can occur at a very early stage. Although the anatomic correction might have been satisfactory over a certain period of time, brutal recurrence can be caused by a violent effort, accompanied by pain. More rarely, the lateral implant fixation is the reason for the recurrence. Uncommon in cases using the transobturator or trans-ligament route, it seems to be a reason for recurrence in techniques using the transmuscular route. We have also observed recurrences after transmuscular posterior IVS. Furthermore (unpublished) studies on cadavers have revealed the poor resistance of levator muscles to traction. This issue has been resolved by using a trans-ligament route at the sacro-spinous ligament. All of these findings support the hypothesis of the possibility of a lateral detachment of the implant, particularly with transmuscular fixation.

Another potential cause of recurrence could be related to individual *anatomic variations* in the bones of the pelvis minor.[52] These variations could influence the positioning of the implants and possibly justify the development of adjustable implants or implants of different sizes.

Decompensation is a mechanism of recurrence that we have already mentioned. As we pointed out in the introduction; compartment correction, especially using a mesh implant, could cause recurrence or decompensation in another compartment. We think that prosthetic repair of a compartment should be considered as an independent risk factor for recurrence in a repair performed on the opposite side. This phenomenon, a known factor for enterocele or rectocele following a Burch procedure or after sacrocolpopexy,[53-55] may be accentuated by overcorrection that sometimes occurs with prosthetic repair or retraction. It is linked to an imbalance in the vaginal axis or an imbalance in the pressures between the different compartments (Fig. 21.11).

This mechanism can be expressed under diverse anatomical structures. As well as "classic" cystoceles and rectoceles, it can make more atypical structures appear. In particular, after posterior prosthetic repair, we have observed cases of anterior colpocele corresponding to an anterior enterocele. Once again, ultrasound facilitates the diagnosis.

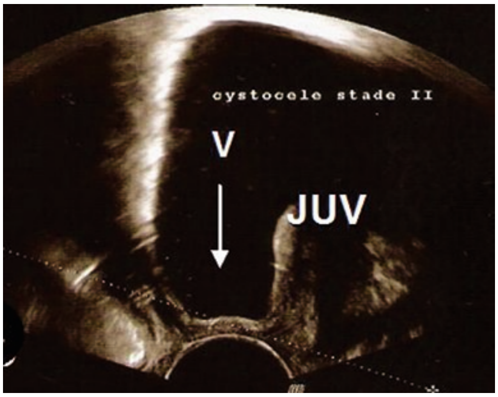


Fig. 21.11 Ba = 0, decompensation of a cystocele after posterior reinforcement (Stage II cystocele V = bladder, JUV = UVJ)

Treatment

Preventive Treatment of Postimplant Recurrence

During prosthetic repair of prolapse, several elements, some based on solid factual arguments and others more hypothetical, can influence the risk of recurrence.

First, there is no clear and concise answer as to the best *moment to operate*. Regardless of functional aspects, the natural history of prolapse remains uncertain. There are conflicting results as to the role the initial stage of prolapse plays as a risk factor for recurrence. The correlation between severity of the initial stage and recurrence reported by some investigators[56,57] was not found by others.[58,59] From an anatomical perspective, there is therefore no certainty as to what point in time an operation presents the least risk of recurrence. Functional criteria do, of course, remain predominant.

A clearer and more precise point is the *choice of implant material*. Since the modest advances obtained using absorbable materials,[32] it seems nowadays that the utilization of nonabsorbable mesh is more effective in reducing recurrence. This assertion is strongly supported by the meta-analysis made by Jia et al.[34] demonstrating a more significant risk for recurrence when absorbable synthetic or biological mesh are used, compared to other nonabsorbable mesh (respectively, OR: 4.12 [IC: 2.2–7.7] and OR: 2.97 [IC: 1.83–4.6]). To date, the use of a nonabsorbable synthetic mesh (preferably woven monofilament polypropylene for better tolerance) is therefore a preventive measure against recurrence.

A much more controversial point is the *prosthetic repair of anatomic compartments other than those indicating the placement of an implant*. For example, what should be done in cases of cystocele arising from a prosthetic repair associated with an asymptomatic retocele, or in the absence of a rectocele? While there is no certainty, two approaches can be considered. The first is abstention (if there is no evidence of prolapse) or nonprosthetic repair (in cases of a non requiring implant concomitant prolapse). This approach is based on the principle of taking preventive measures to reduce the risk of complications. It is considered by some, and we share this view, that in certain situations, the risk of decompensation is such that it is preferable to place an implant in one compartment if another implant has been placed opposite. In our opinion, this needs to be discussed in the following situations:

- As an indication in rectocele repair using an implant, with stage 2 cystocele: we suggest adding an anterior implant,
- In cases of severe multicompartmental prolapse: a complete prosthetic repair,
- As an indication in cystocele repair using an implant, with Stage 2 rectocele: nonsystematic posterior implant, depending notably on the risk factors.

The best anatomic outcomes obtained by Elmer et al.[60] concomitantly using anterior and posterior implants compared to the isolated procedure using the TVM technique supports this synergic or more balanced view of complete repairs (anterior AND posterior) compared to unicompartmental prosthetic repairs.

In the specific case of sacrocolpopexy, there is less controversy surrounding the issue. Most authors systematically place a posterior implant.[31,61] This practice, facilitated by laparoscopic monitoring, is used as a result of the incidence of rectoceles occurring after sacrocolpopexy without posterior implant placement.[31,54] Some authors, however, attribute the occurrence of these rectoceles to the concomitant practice of performing a Burch procedure rather than to the actual sacrocolpopexy and advocate a posterior prosthesis only in cases of proven rectocele, notably due to the increased risk of constipation, dyspareunia, and rectal injury.[54]

The concept of complete repair developed by abdominal route has influenced thinking on multiple prosthetic repairs by vaginal route.

The mechanism of the *preventive role of suburethral tapes* in the recurrence of cystocele has already been discussed above. We emphasized the ambiguity between recurrence of cystocele and urethro-trigonocele. It is probably this ambiguity that explains the effect, observed by several authors, of suburethral or subcervical sling placement[32] on the recurrence of cystocele.[62] The precise role of this type of procedure in preventing or decreasing recurrence is yet to be determined. The decision to place a suburethral tape is currently based on the associated urinary symptoms.[63]

The type of *mesh reinforcement technique chosen* probably influences the risk of recurrence. We were not, however, aware, at the date of publication, of any comparative study between any two mesh repair techniques for prolapse. Neither, unfortunately, are there any more studies comparing transabdominal and transvaginal mesh repair than comparisons between the different transvaginal prosthetic repairs. The only arguments we can put forward, therefore, are indirect ones.

Sacrocolpopexy reduces the risk of recurrence compared to transvaginal sacrospinous fixation.[16] On the other hand, there is no direct comparison between sacrocolpopexy and equivalent transvaginal prosthetic repair. However, the results of recent studies, published in a review of the literature,[64] show similar anatomical outcomes between transvaginal prosthetic repair and sacrocolpopexy.

Alternative transabdominal prosthetic repairs have been described[29] with good outcomes, but need more in-depth evaluation, particularly compared to sacrocolpopexy.

The large number of transvaginal techniques means that analysis is complex. We will simply give a description, without being able to draw any conclusion, of various complete repair techniques using implant placement in several compartments. The outcomes cited as an example refer only to prospective studies, but should be considered with caution, given the wide variety of procedures that are to be found in each study. The results are given in Table 21.3 and include the proportion of patients having undergone a mesh repair on several compartments.

These findings are too heterogeneous to be able to come to any firm conclusions as to methods which can prevent recurrence. Only well-conducted comparative studies will show which techniques should be preferred or provide preferential indications.

The *role that a hysterectomy plays* in the prevention of recurrence is the subject of controversy. It is ineffective in improving anatomic outcomes in transabdominal repair and has a deleterious effect on erosion rates in cases of total hysterectomy.[30] Preserving the uterus or subtotal hysterectomy is the rule in sacrocolpopexy procedures. This approach has no effect on recurrence. The debate surrounding vaginal implant surgery was rekindled by the link revealed between a hysterectomy and implant exposure.[40] As regards recurrence, we observed, in a multicentre retrospective study of 110 Prolift® procedures, two cases of uterine relapse.[74] These cases occurred at the time we started using the technique, when absorbable thread was used for uterine fixation to the mesh. Uterine preservation must therefore be concomitant with careful assessment of the degree of uterine prolapse and, if necessary, suitable fixation.

Curative Treatment of Postimplant Recurrence

We start off by discussing the general principles that should guide the management of recurrence after implant placement. We then examine certain clinical situations of recurrence, on

Table 21.3 Multicompartmental transvaginal mesh repair techniques

Reference ("population" at "mean follow-up")	Technique	Material	Total prosthetic repair rate	Recurrence
Altman et al. 126 at 2 months)[65]	Insertion Kit and transobturator and transligament passage	Monofilament polypropylene (Prolift®)	25%	$C = 13\%$ $R = 9\%$ $\surd = /$
Doumerc et al. (132 at 21 months[66]	Implant 4 × 7 cm fixations with sutures	Pelvicol®	63.6%	$C = 16.8\%$ $R = 8.4\%$ $\surd = 16.9\%$
Flam (55 at 3 months)[67]	Insertion Kit and transobturator and transligament passage	Monofilament polypropylene (Prolift®)	7%	$C = 0$ $R = 1.8\%$ $\surd = /$
De Tayrac et al. (143 at 13 months)[68]	Variable	Monofilament polypropylene (Ugytex®)	45.5%	$C = 6.8\%$ $R = 2.6\%$ $\surd = /$
Agarwala et al. (39 at 24 months)[69]	Colpocleisis (Lefort operation)and excess prosthetic fragments in detachment	Porcine intestinal submucosa 28% (Xenograft Stratasis®) 72%monofilament polypropylene (TVT®)	?	$C = 2.5\%$ $R = 2.5\%$ $\surd = 5\%$
Sergent et al. (103 at 32 months)[70]	"Transobturator Infracoccygeal hammock": T-shaped prosthesis with 2 transobturator arms and posterior IVS attachment	Polyester (Parietex®): 21% Multifilament polypropylene (Surgipro Mesh®): 25% Collagen-impregnated monofilament polypropylene (Ugytex®): 53%	100%	$C = /$ $R = 2\%$ $\surd = 3\%$
Foulques (317 followed-up: 85% at 3 months, 61% at 1 year, 25% at 3 years, 19% at 4 years)[71]	Customized and lateral fixation	Monofilament polypropylene (Gynemesh® and Gynemesh® PS)	75%	$C = 4.1\%$ $R = 1.9\%$ $\surd = 6\%$
Carey et al. (95 followed-up 84% at 1 year 72)	Customized, distinct anterior and posterior prosthesis, unanchored lateral arms, postop pessary	Monofilament polypropylene (Gynemesh PS ®)	66.3%	$C = 10\%$ $R = 6.2\%$ $\surd = 15\%$
Milani et al. (71 at 9 months)[73]	Customized, distinct anterior and posterior prosthesis, unanchored lateral arms, postop pessary	Titanium-impregnated Polypropylene (Ti-Mesh ®)	14.3%	$C = 36\%$ $R = 18\%$ $\surd = /$

C recurrence of cystocele, *R* recurrence of rectocele, recurrence of at least one compartment a case-by-case basis, while proposing a number of reasoned therapeutic choices.

As most recurrences are asymptomatic, a number of teams describe surgical abstention. Follow-up of moderate and/or asymptomatic recurrences is important in tracking their development and in determining the risk factors that could lead to surgical treatment.

As we saw before, recurrence can be associated with retraction. As the potential link to other complications, such as implant exposure or organ erosion, remains an unknown quantity, we consider that the possibility of an associated complication should always be investigated in the event of a recurrence. The clinical examination needs to be particularly meticulous, with the routine use of cystoscopy in cases of anterior implants. In cases of posterior implants with rectal symptoms, a proctoscopy should also be considered. The type of recurrence and its mechanism can be determined through ultrasound analysis. We consider that this preliminary analysis is an essential part of the treatment, which has to factor in the mechanism responsible for the recurrence.

The therapeutic principles will therefore vary depending on whether this mechanism results from a decompensation, a recurrence through lack of coverage, or a fixation defect.

In cases of decompensation, it should be taken into account that the compartment treated by implant placement cannot balance the pressures exerted on the decompensated compartment and that repair at this level must be particularly efficacious. If decompensation is related to a cystocele (*following prosthetic treatment of a rectocele*), we usually opt for implant repair, as cystocele is a high-risk situation for recurrence. If decompensation is related to a rectocele (*following prosthetic treatment of a cystocele*), we opt for a conventional repair if no other procedure had been previously performed, and for an implant if a nonprosthetic rectocele repair was initially performed or if the rectocele is very voluminous. When there are associated risk factors of recurrence, a prosthetic repair is indicated.

In cases of lack of coverage, the strategy varies, depending on whether this lack of coverage is due to an undersized implant or an implant that was initially sufficiently large but which subsequently retracted. When the initial prosthesis is too small, recurrence can be exactly the same type as the initial prolapse and a larger implant is indicated. When the implant is retracted (or in certain cases when the implant is too small), the prolapse occurs, as we saw before, on the edge of the implant, in the noncovered area. The recurrence occurs through invagination or in an atypical location. In such a case, how can the weak point be covered and what can be done with the retracted area? If the retraction is symptomatic or particularly significant, it would be better at first to resect either the part concerned or the whole of the implant. This is a fairly delicate surgical act, which, particularly if the vagina is of poor quality or extensively resected, can cause the repair of the recurrence to be deferred after scar formation. If there remains a well-tolerated implant fragment, it can be used as an anchoring point for the new implant. For example, in cases of vaginal cystocele, if the defect is not too large, a TVT or TOT tape can be placed on the low cystocele. This technique was successfully described in a small study of recurrences of cystocele after nonprosthetic repair.[75] In cases of enterocele through invagination between an anterior and a posterior implant, once the enterocele sac is resected, is it advisable to locate the two prosthetic edges and anchor them using nonabsorbable sutures in order to eliminate the weak point.

In a study involving 19 cases of implant complications referred to their department, Ridgeway et al.[76] reported six cases of recurrence. In all recurrences, the existing implant was resectioned and the recurrence was treated without a new implant. Of the 19 patients half were reoperated for recurrence.

Recurrence after sacrocolpopexy occurs mostly as a result of lack of coverage of a vaginal rectocele. Prosthetic or nonprosthetic repair can be performed using the vaginal route. A laparoscopic approach may also be used. A voluminous enterocele or a voluminous high rectocele may require an implant. Cystoceles following sacrocolpopexy occur either under the lower edge of an implant which has insufficiently been pulled down or which has retracted upwards, or, most commonly, through a paravaginal defect. Most often, we correct this defect by vaginal route with an implant with transobturator arms.

In cases of fixation defects, the simplest case is uterine fixation laxity. The commonest example is when the fixation of the uterus to the prosthesis loses its tightness. This type of recurrence, which can happen early or suddenly, can be treated by resecuring the uterus to the implant, possibly concomitantly with the amputation of a hypertrophied cervix. Another option is to perform a hysterectomy if there are strong anterior and posterior implants that will then be anchored together in order to close the space and prevent a potential enterocele. Performing a transvaginal hysterectomy after prosthetic repair is a relatively simple technique, in spite of initial reservations. In a study of 110 Prolift® procedures where two uterine relapses were observed, one was treated by vaginal hysterectomy and the other by sacrocolpopexy.[74]

In cases of fixation defects due to an undersize implant (after repair using a "four-corner" kind technique), our main aim is to place a larger implant.

Conclusion

Recurrence following implant placement is still poorly understood both as regards its exact incidence and its complex physiopathology. For this reason, considerable work is required to determine the most effective means to further reduce it.

Nevertheless, we already possess sufficient experience for proper management. This must be accompanied by a meticulous clinical examination and is considerably facilitated by ultrasound.

From a surgical perspective, solutions do exist. They are based upon a few general principles and a case-by-case approach. After managing a potential associated complication, there is no standard procedure for repairing a defect, but a new implant is often required. A careful follow-up of our patients will certainly provide us with the best solutions.

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22

Postoperative Infections in Pelvic Reconstructive Surgery

Sebastian Faro

Introduction

Postoperative infection continues to be a problem and although measures are taken to reduce the incidence of postoperative infection, the morbidity and mortality are of significant concern. The Centers for Disease Control and Prevention (CDC) reported in 1999 that there were 30 million operations performed each year.[1,2] The actual or true rate of surgical site infection (SSI) are not known, because many are not reported and many occur after the patient is discharged from the hospital. The reported rates of SSI range from 2% to 3%, but this is just an estimate; the actual rate may approach 750,000 and approximately 500,000 may be limited to the incision.[3,4] The Department of Veterans Affairs monitored SSI for the last 20 years and reported an SSI rate of 5.1%, compared to 3.6% for pneumonia, Urinary tract infection 3.5%, and systemic sepsis, 2.1%.[5] In 2002, in the USA, there were approximately 14 million operative procedures that resulted in a nosocomial infection rate of 17%.[6] The four main infections that resulted in death (98,987) were pneumonia (35,967), SSIs (8,205), urinary tract infection (13,088), and bacteremia (30,665).[6] Nosocomial infection or health-care-associated infections (HAI) are a common cause of morbidity and mortality in the USA and are frequent, if not the most common, adverse events associated with health care.[7] The CDC classifies SSIs into three categories: (1) superficial incisional involving the skin and subcutaneous adipose tissue, (2) deep incisional involving fascia and muscle, and (3) involving an organ and intraperitoneal spaces.[1]

Hysterectomy continues to be a common operative procedure with 3.1 million hysterectomies performed in the USA from 2000 through 2004.[8] The rate of hysterectomy was highest among women of age 40–44 and lowest among women of age 15–24.[8] The three most common conditions associated with hysterectomy were uterine leiomyoma, endometriosis, and uterine prolapse.[8] Hysterectomy, whether performed abdominally, vaginally, or laparoscopically, is a clean contaminated surgical procedure. This fact places the patient undergoing hysterectomy at significant risk for the development of postoperative pelvic infection. Several studies have attempted to demonstrate that fever alone is not a valid indicator of infection.[9–11] It is sometimes difficult to distinguish between fever not associated with infection and fever indicative of infection. This is true when relying on fever alone as there are many factors known and unknown that can give rise to fever in the postoperative patient, e.g., anesthetic agents, trauma of surgery, medications administered postoperatively (drug fever). Fever not associated with infection does not have a specific pattern, but fever associated with infection is typically accompanied by a tachycardia. The fever and pulse rate, in the presence of infection parallel each other. Attempting to assess the patient for the presence of a postoperative SSI can be difficult if one relies on one clinical criterion, such as fever. A proper assessment of the patient suspected of having a postoperative SSI following vaginal surgery depends on the presence of fever plus tachycardia and the findings of the physical examination including the pelvic examination. These results should lead the physician to ordering the proper tests.

Postoperative infection associated with pelvic reconstructive surgery can involve one site or involves multiple sites. The concept of surgical site in pelvic reconstructive surgery is very different from that of the patient undergoing a laparotomy for bowel surgery or a thoracotomy. The patient undergoing a vaginal hysterectomy has one surgical site when the circumferential incision is made in the cervix. However, when the attachments to the cervix and uterus are severed, these sites must also be considered as surgical sites, because each of these, especially the fallopian tubes and pelvic peritoneum are at risk for infection. If additional procedures are performed, for example, anterior and posterior colporrhaphy, or a suburethral incision are all additional *surgical* sites. Therefore, in pelvic reconstructive surgery, the patient has multiple surgical sites and there is a significant risk for postoperative pelvic infection. The addition of synthetic mesh to re-enforce the

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weaken collagen tissue in these compartments may add a significant additional risk factor for the development of postoperative infection. In patients undergoing vaginal hysterectomy, postoperative pelvic infections are mostly (probably 95%) due to the patient's own endogenous vaginal bacteriology.[12]

Microbiology

The microbiology of the lower genital tract is complex and is made up of numerous gram-positive and gram-negative facultative and obligate anaerobes (Table 22.1). The microenvironment of the vagina is maintained in a very delicate balance. The equilibrium of the vaginal ecosystem can be easily disrupted by a variety of mechanisms, e.g., antibiotics for treating infections distant from the vagina, inappropriate use of antimicrobial agents to treat various conditions of the vagina many of which may be thought to be of an infectious origin but are not. Hormonal treatment can impact the microbial growth of the vagina; this is exemplified by the postmenopausal women not on hormones whose vaginal microflora becomes dominated by gram-negative facultative anaerobic bacteria. The frequency of sexual intercourse can also impact the vaginal ecosystem via the alkaline semen which can cause the pH of the vagina to destabilize and achieve hydrogen ion concentrations that render the pH > 5. Disruption in the vaginal bacteriology can place the patient at risk for postoperative pelvic infection. One of the main factors in maintaining the equilibrium of the vaginal ecosystem and balanced endogenous vaginal microflora is the pH (Fig. 22.1). Lactobacillus species are considered to be the dominant bacterium in women whose vaginal ecosystem is in balance.[13] A pH between ≥ 3.8 and ≤ 4.5 is required for *Lactobacillus crispatus* or *L. casei* or *L. jensenii* to maintain dominance. *Lactobacillus* maintains dominance through, at the least, three mechanisms: production of organic acids, mainly lactic acid, hydrogen peroxide, and a bacteriocin named lactocin.[14] One well-known mechanism of bacterial antagonism whereby one bacterium can inhibit the growth of another bacterium is via the production of hydrogen peroxide (H_2O_2).[15–17] Certain species of *Lactobacillus* utilizing flavoproteins convert oxygen to hydrogen peroxide and because they lack heme catalase, H_2O_2 accumulates and is secreted into the bacterial environment.[18] Myloperoxidase forms a complex with halides and H_2O_2 which is toxic to bacteria, e.g., *Escherichia coli*, *Gardnerella vaginalis*, and obligate anaerobes.[17]

Table 22.1 Bacteriology of the vagina

Gram-positive facultative anaerobes	Gram-negative facultative anaerobes
Lactobacillus crispatus	*Enterobacter aerogenes*
L. casei	*E. agglomerans*
L. jensenii	*E. cloacae*
Corynebacterium	*Escherichia coli*
Staphylococcus aureus	*Klebsiella pneumoniae*
Staphylococcus epidermidis	*K. oxytoca*
Enterococcus faecalis	*Morganella morganii*
	Proteus mirabilis
	P. vulgaris
Gram-positive obligate anaerobes	Gram-negative obligate anaerobes
Atophobium vaginae	
Peptococcus niger	
Peptostreptococcus anaerobius	*Fusobacterium necrophorum*
	Fusobacterium nucleatum
	Mobiluncus sp.
	Prevotella bivia
	P. disiens

Lactobacillus lactocin is a low molecular weight protein that inhibits a variety of gram-positive and gram-negative aerobic, facultative, and obligate anaerobic bacteria.[19] Lactocin is produced by many strains of *Lactobacillus*.[14,19] Bacterocins, e.g., lactocin, like antibiotics inhibit bacteria growth in manner similar to antibiotics but differ on the basis of their synthesis, mechanism of action, toxicity, and resistance mechanisms.[20,21] Similar to some antibiotics, bacteriocin disrupts the bacterial cellular membrane ($\Delta\psi$ dissipation) and induces ATP efflux because of pore formation.[22]

The endogenous bacteriology of the lower genital tract contains many pathogenic bacteria. When the endogenous vaginal bacterial community is dominated by the appropriate species of *Lactobacillus*, e.g., *L. crispatus*, *L. casei*, or *L. jensenii*, the pH of the environment is maintained between 3.8 and 4.5 and the growth of the pathogenic bacteria is suppressed (Fig. 22.1). If *G. vaginalis* assumes dominance during the transition phase, growth of *G. vaginalis* will continue to lower the oxygen concentration and increase the pH creating an environment that favors growth of obligate anaerobes. The gram-negative facultative bacteria will switch from an aerobic to an anaerobic metabolism (Figs. 22.2 and 22.3). This environment changes the ratio of *Lactobacillus* to pathogenic bacteria in favor of the latter. Thus, the patient undergoing pelvic reconstructive surgery, whose vaginal microflora is dominated by a gram-negative facultative anaerobe (e.g., *E. coli*) or obligate anaerobes (BV), is a significant risk for developing a postoperative infection. There are approximately 600,000 hysterectomies performed annually in the USA and approximately 150,000 (25%) are performed vaginally.[23] Based on the number of vaginal hysterectomies, the number of estimated wound infections is 3,150–14,500.[24–26] The actual incidence of infection when

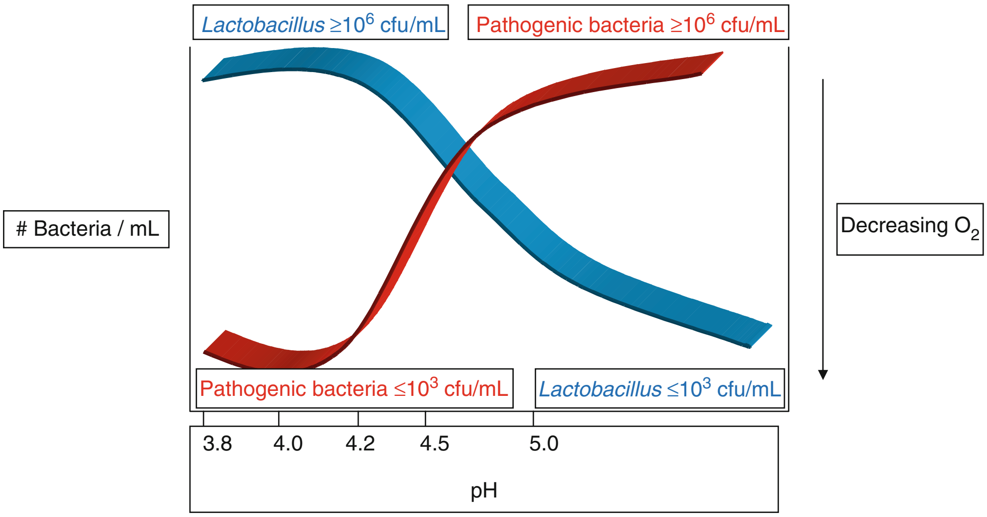


Fig. 22.1 Graph depicts changes if pH reflects changes in growth of *Lactobacillus* and pathogenic bacteria. The pH between 4.5 and 5 is a transition zone where, depending on which bacterium assumes dominance will determine the composition of the vaginal microflora. The importance of the pH reflects the size of the bacterial inoculum with regard to composition of the bacterial community. When the pH is <4.5, *Lactobacillus* will ≥1,000,0000 bacteria/mL of vaginal fluid and the pathogens will ≤1,000 bacteria/mL of vaginal fluid; this represents a ratio 1,000:1

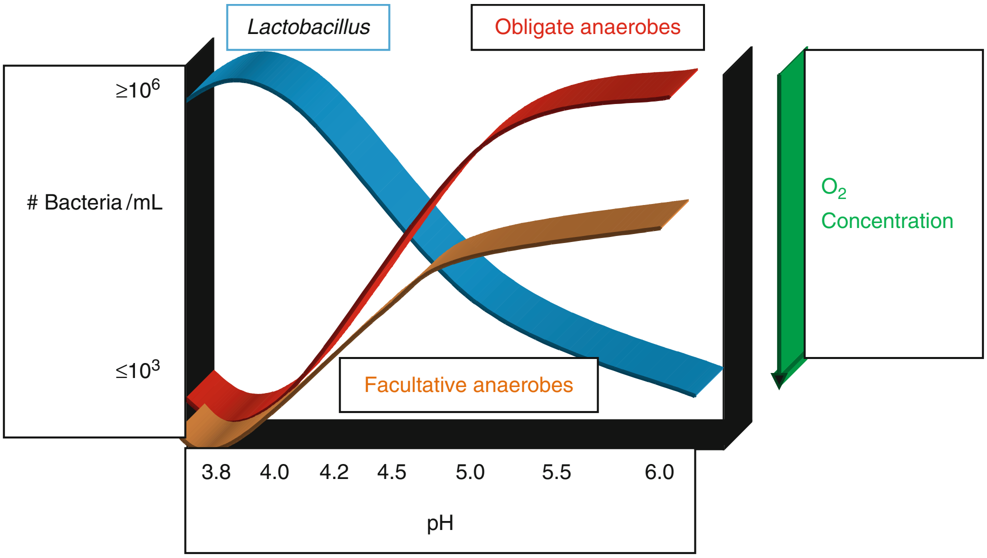


Fig. 22.2 The decrease in O_2 concentration impacts which organisms achieve dominance. In this graphic depiction, depletion of oxygen results in increased growth of obligate anaerobes. The facultative anaerobic bacteria switch from an aerobic to anaerobic metabolism; they achieve a concentration that is $\geq 10^5$ bacteria/mL of vaginal fluid. This graph is an example of the evolution of BV and represents a tremendous inoculum, if the patient is undergoing vaginal surgery

combining vaginal hysterectomy with pelvic reconstructive surgery for prolapse may be higher, and when considering pelvic reconstructive surgery in the absence of hysterectomy the infection rate may be lower. However, the placement of synthetic mesh may result in a significant number of infections based on the number of repairs performed in the patient.

The vaginal endogenous pathogens consist of a large number of gram-positive and gram-negative facultative and obligate anaerobes. When the obligate anaerobic bacteria are dominant, facultative anaerobes are present in significant numbers, too. When the numbers of facultative anaerobic bacteria are present in a concentration $\geq 10^5$ bacteria/mL, the risk of infection is increased. When the numbers of both facultative and obligate anaerobic bacteria are present in the same environment, and concentration $\geq 10^5$ bacteria/mL, there is a significantly increased risk for infection. Combinations of bacteria may grow synergistically to form abscesses; this has been demonstrated in the rat. Onderdonk et al. demonstrated that the initial infection begins as a peritonitis and the subsequent abscesses involved *Bacteroides fragilis*, *Fusobacterium*, *E. coli,* and *Enterococci*.[27] Martens et al. demonstrated the formation of intra-abdominal abscesses in rats using clinical isolates of *Enterococcus faecalis*, *E. coli*, and *B. fragilis* obtained from human vaginas.[28]

Thus, the endogenous bacteria of the vagina contain the pathogens responsible for most of the infections that occur following vaginal surgery. Studies have demonstrated that patients undergoing hysterectomy have an increased risk of

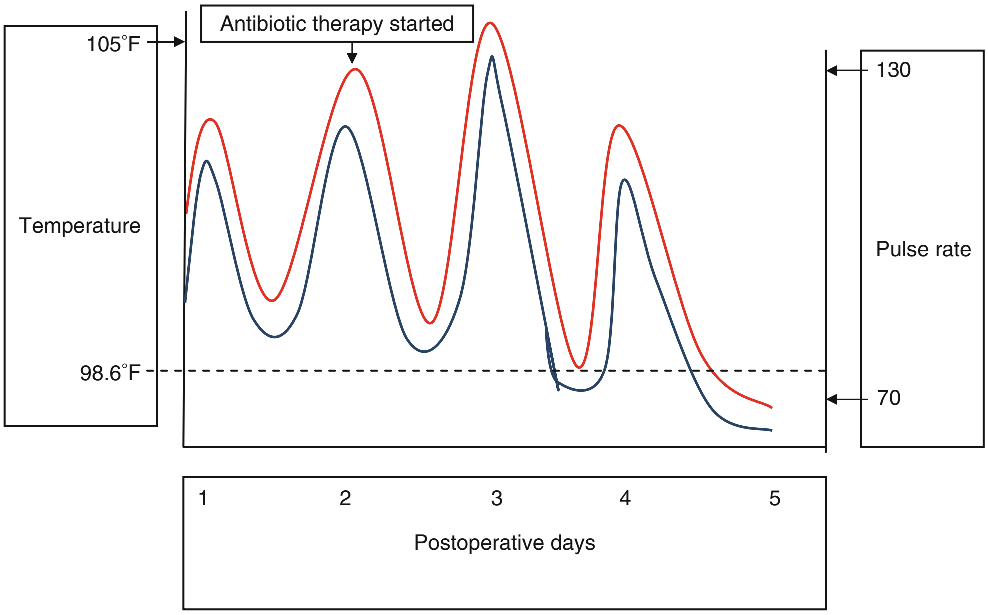


Fig. 22.3 Depiction demonstrating parallel course of fever and pulse curves. Note that the zenith in pulse rate corresponds to peak in temperature

infection if they have bacterial vaginosis (BV). Lin et al. demonstrated that BV was associated with postoperative infection.[29] These investigators also found that patients with a transitional vaginal microflora compare to patients with a *Lactobacillus* dominant vaginal microflora and were not at significant risk for postoperative infection.[29] The association between BV and postoperative infection has been reported by other investigators.[30,31] Larsson and Carlsson demonstrated that preoperative and postoperative treatment of patients undergoing abdominal hysterectomy with metronidazole reduced the incidence of vaginal cuff infection in patients with an abnormal vaginal microflora.[32] The current author conducted a study (S. Faro, M.D., unpublished data, June 2010), where the vaginal pH was determined immediately prior to the vaginal prep in patients undergoing vaginal and abdominal hysterectomy. All patients received cefazolin 1 g IV for surgical prophylaxis. There were 83 patients whose vaginal pH < 4.5 and did not develop a postoperative pelvic infection; there were 12 patients whose vaginal pH $>$ 4.5 but <5 who developed febrile morbidity but did not develop a postoperative infection. There was a total of five patients whose vaginal pH > 5who did develop a postoperative infection. Febrile morbidity is defined as an elevated body temperature ≥100.4°F (38°C) measure on two separate occasions taken at least 4 h apart and a normal pulse rate (<90 beats/min).

The available data indicates that patient with an abnormal vaginal microflora, especially BV, but not limited to BV are at considerable risk for the development of postoperative infection if undergoing pelvic surgery. With regard to the vaginal microflora, the key points are: (1) an abnormal vaginal flora, e.g., BV or microflora dominated by gram-negative facultative bacteria; (2) vaginal microflora dominated by *Streptococcus agalactiae* in the case of patients with significant chronic illness; and (3) exposure of the deep vaginal tissues to a non-*Lactobacillus* dominated microflora. The key is recognizing that a non-*Lactobacillus* dominant microflora is indicative of a vaginal microflora dominated by pathogenic bacteria. The inoculum size is well beyond the numbers required for infection. Infection is preceded by bacterial contamination and surgery performed through the vagina is operating in a contaminated field. Contamination does not mean that infection is inevitable, but the risk for infection is dependent upon the host defenses, the trauma to the tissues, and the numbers of bacteria present at the operative site, i.e., the bacterial inoculum size. The required inoculum for surgical site infection is between $>10^5$ and $>10^4$ bacteria/g tissue.[33,34]The inoculum size in relation to the endogenous vaginal microflora far exceeds the required numbers of bacteria to induce infection. A second contributory factor in initiating infection at a surgical site is the presence of foreign material. Elek and Aness demonstrated that an inoculum of 10^6 bacteria/g of tissue was required to initiate an infection de novo.[35] When a foreign body, e.g., silk suture, was placed in the wound, the required inoculum size was decreased to 10^3 bacteria/g of tissue.[35] Surgical procedures conducted through the vagina in the presence of an abnormal endogenous vaginal microflora meet the required inoculum size for the development of infection. Foreign bodies are present, i.e., suture material and the presence of synthetic mesh which enhance the risk of infection.

Postoperative Infection

Clinical Presentation

There are a variety of infections that are associated with pelvic reconstructive surgery (Table 22.2). The clinical indications of postoperative infection are fever, tachycardia, and elevated white count. The physical findings of postoperative pelvic infection are purulent discharge which may or may not be present, edema at the surgical site, pain not responsive to appropriate pain medication. Patients who develop fever in the absence of a tachycardia typically are not infected. Fever in the absence of tachycardia may be exhibiting a response to cytokine release secondary to tissue destruction associated with the surgical procedure, or perhaps the presence of the graft or suture material, perhaps if vicryl + suture is used, Triclosan (antiseptic) is used to coat the suture which may cause a reaction, and various medications including antibiotics.

Fever is defined as an oral temperature of ≥100.4°F measured on two separate occasions at least 6 h apart or a temperature ≥101°F occurring at any time. Fever as indicator for postoperative infection is a poor indicator when used alone. Fever following laparotomy is a common occurrence, and has been reported in 5–75% of patients.[36,37] De la Torre et al. demonstrated that fever in association with an elevated WBC count in patients who had surgery for gynecologic malignancy, bowel resection, number of febrile days, higher fever, was more likely to be consistent with the presence of infection.[11] There are several studies attempting to use fever solely as a potential marker or indicator for the presence of postoperative infection.[38,39] There should be a tachycardia that parallels the temperature course (Fig. 22.3). When these two events occur simultaneously, the patient should be evaluated to determine if an infection is present. Failure to initiate an evaluation in a postoperative patient with a temperature elevation (≥100.4°F measured on two occasions at least 6 h apart, or ≥101°F at any time) with a concomitant tachycardia (Pulse rate ≥ 90 beats/min) can result in a serious infection, e.g., sepsis, septic shock, or necrotizing fasciitis. Table 22.3 depicts the basic work-up for a patient suspected of having a postoperative infection. There is no specific time requirement for the evolution of a postoperative infection. The time at which an infection makes itself known is dependent upon the bacterium or bacteria involved. A bacterium such as *Streptococcus pyogenes* (group A streptococci, GAS) can reveal its presence very early in the postoperative period, especially if it is a toxogenic strain. Many of the gram-negative facultative anaerobic bacteria inhabiting the lower genital tract are extremely virulent. The gram-positive and gram-negative facultative anaerobic bacteria reproduce approximately every 30 min and the obligate anaerobic bacteria reproduce approximately every 4 h. Therefore, a patient having vaginal surgery, whose endogenous vaginal microflora is dominated by a variety of pathogenic bacteria already has an extremely large number of bacteria present at the operative site (Fig. 22.4). The number of bacteria in the vagina when there is an imbalance in microflora can reach ≥10^8 bacteria/mL of vaginal fluid. This number of bacteria in association with tissue hypoxia, collection of blood in the operative site, plus foreign bodies (suture and graft material) is all the ingredients for the development of infection.

Table 22.2 Infections associated with pelvic reconstructive surgery

Infections associated with pelvic reconstructive surgery: (a) Vulva infection – associated with trans-obdurator sling, cellulitis, abscess, biofilm of the graft (b) Anterior vaginal compartment – cellulitis, abscess, graft biofilm (c) Posterior vaginal compartment – cellulitis, abscess, graft biofilm (d) Vaginal hysterectomy – cellulitis, abscess
Infections associated with pelvic reconstructive surgery but distant from the surgical site: (a) Urinary tract infection – cystitis, pyelonephritis (b) Bacteremia (c) Pneumonia (d) Sepsis (e) Septic shock (f) Necrotizing fasciitis

Table 22.3 Antibiotic choices for empiric therapy for the treatment of postoperative pelvic infection

Antibiotic	Bacterial spectrum of activity
1. Piperacillin/tazobactam (Zosyn)	Gram-positive and gram-negative facultative and obligate anaerobes
2. Ertapenem (Invanz)	Gram-negative facultative and obligate anaerobesWeakness *Enterococcus*, *Pseudomonas*
3. Clindamycin	Gram-negative and gram-positive obligate anaerobes *Streptococcus agalactiae* ~20% resistance Methicillin-resistant *Staphylococcus aureus* ~15–20% resistance No activity against *Enterococcus* and gram-negative facultative anaerobes
4. Metronidazole	Active only against gram-positive and gram-negative obligate anaerobes
5. Aminoglycosides	Gram-negative facultative anaerobes Methicillin-resistant *Staphylococcus aureus*

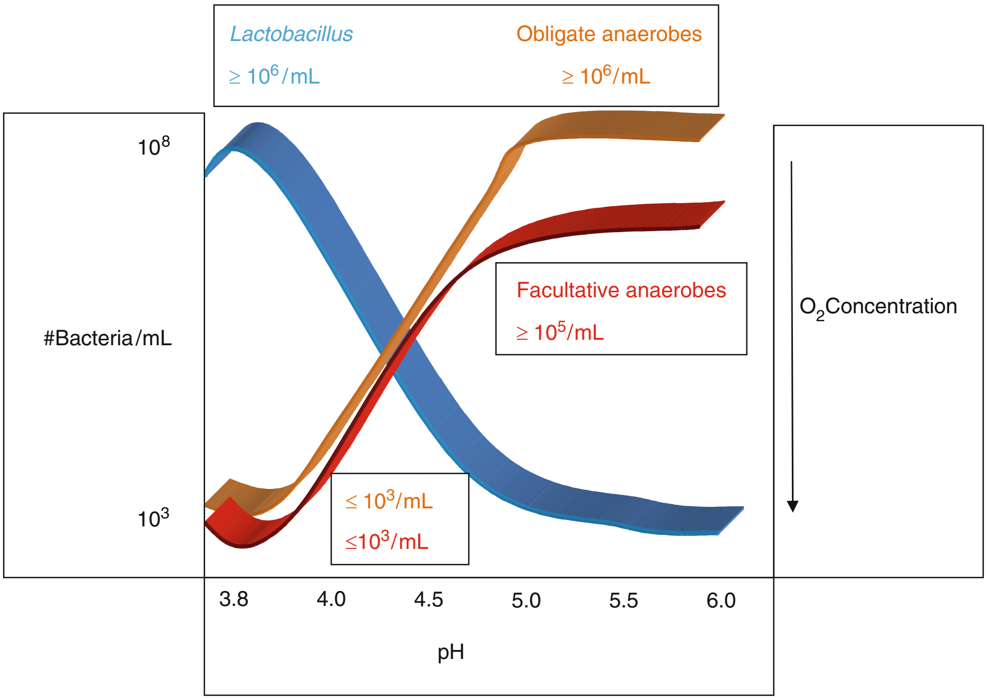


Fig. 22.4 Note that when *Lactobacillus* is dominant the ratio of *Lactobacillus* to pathogens is 1,000,000:1,000 or 1,000:1. Therefore, the inoculum of pathogens is insufficient to initiate infection. When *Lactobacillus* looses dominance the ration becomes reversed, i.e., lactobacilli : pathogens is 1,000:1,000,000 or 1,000:1. The number of pathogenic bacteria or inoculum is more than sufficient to initiate infection, especially if there are contributing factors present at the surgical site

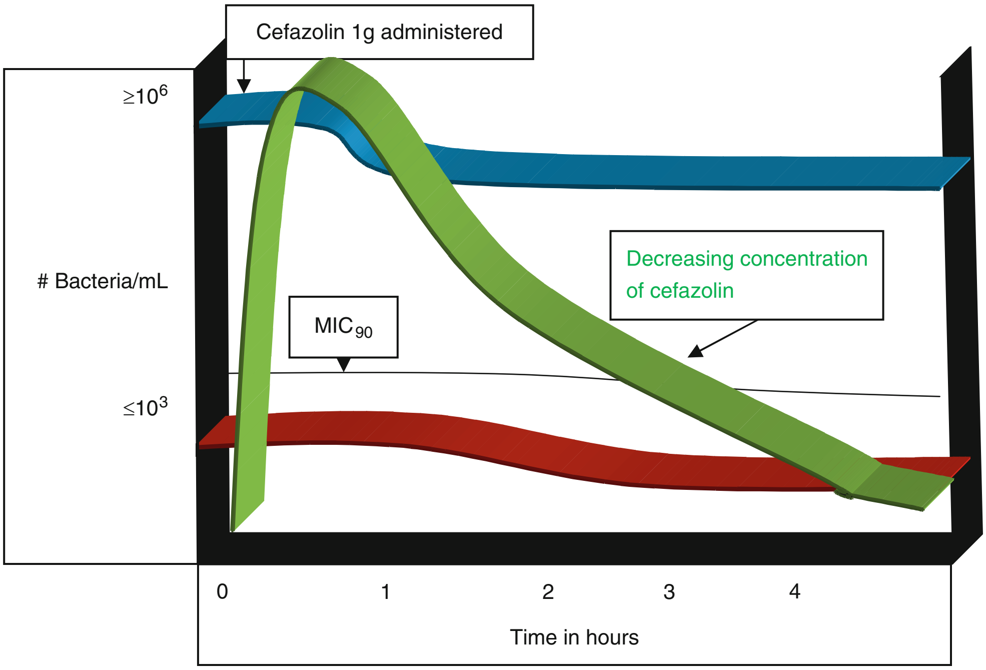
Prevention of Postoperative Pelvic Infections

Data regarding the use of prophylactic antibiotics administered for the prevention of postoperative infections in patients undergoing vaginal reconstructive surgery are not abundant. However, there are a great deal of data available in both the obstetric and gynecologic literature to draw upon. The data have demonstrated that antibiotics administered within 30–60 min preceding making the incision significantly reduces postoperative infection in the obstetric and gynecologic patients.[40–43] Cefazolin has continued to be the most frequently used antibiotic for surgical prophylaxis in patients undergoing cesarean section and for patients undergoing abdominal or vaginal hysterectomy. Other antibiotics have also been shown to be effective for surgical prophylaxis, but because cefazolin is inexpensive and effective, it continues to be frequently administered for surgical prophylaxis. Patients with an altered vaginal microflora, especially those with bacterial vaginosis are most likely not to benefit from antibiotic administration for surgical prophylaxis and develop a postoperative pelvic infection.[29] The risk of postoperative pelvic infection is lowest in those patients with a *Lactobacillus*-dominant endogenous vaginal microflora. Therefore, it would be most prudent to screen the patient prior to surgery to determine the status of the endogenous vaginal microflora, and if altered, treat the patient in an attempt to restore *Lactobacillus* to dominance, thereby reducing the inoculum size of the endogenous pathogenic bacteria and allowing the antibiotic to further suppress the growth and survival of the pathogenic bacteria (Fig. 22.5). When the number of pathogenic bacteria far outnumbers the number of lactobacilli, e.g., BV or gram-negative facultative anaerobic dominant microflora, the prophylactic antibiotic dosage is not sufficient to overcome the inoculum and infection results. This is the most likely explanation for the failure of prophylactic antibiotics to prevent postoperative pelvic infection in healthy patients.

Clinical Presentation and Diagnosis

The patient with a postoperative pelvic infection presents with an elevated oral body temperature, tachycardia, and elevated WBC count.[42,44] To reiterate, fever in the absence of tachycardia is most likely not indicative of infection. The concomitant presence of an elevated WBC count should initiate examination of the patient. A pelvic examination should be performed to determine if there is increased temperature at the vaginal apex and pain on palpation. This would indicate the presence of cellulitis and infection. In addition to obtaining a WBC count with differential, serum electrolytes, blood urea nitrogen (BUN), serum creatinine, and glucose should also be obtained. If the patient is elderly, a manual white cell differential should be obtained, because elderly patients may not manifest a significant rise in the total WBC count but can show an increase in immature neutrophils (Bands). Greater than 10% increase in immature neutrophils

Fig. 22.5 The concentration of cefazolin in serum and tissue decreases over time; at 3 h infusion, the concentration will be below the MIC_{90} of the pathogenic bacteria found in the vagina. Therefore, the antibiotic must be at maximum concentration at the time the incision is made and remain above the MIC_{90} for at least 3 h. If the operation lasts longer than 3 h a second dose should be administered



or bands is indicative of an inflammatory response secondary to infection. Blood glucose determination is important, because a value ≥200 mg/dL raises the patient risk for the development of infection. The BUN and creatinine are necessary, because if beta-lactam antibiotics, aminioglycosides, and carbapenems are administered, these antibiotics are excreted by the kidneys. If the patient's kidney function is compromised, then these antibiotics will require adjustment in dosage or change in interval between doses.

The pelvic examination is important for several reasons: (1) to determine if cellulitis is present, (2) if there is rebound on the bimanual examination this would indicate the presence of pelvic peritonitis, (3) to determine if a mass is present, and (4) to determine if there is drainage issuing from the vaginal suture line. Auscultation of the abdomen, especially the lower abdomen, will reveal if bowel sounds are present or absent. If bowel sounds are absent in the lower abdomen and the patient has pelvic cellulitis this finding suggests the presence of an ileus. The presence of an ileus in the lower abdomen in patients with pelvic cellulitis should be considered as a significant infection. The ileus can spread to the upper bowel in the upper abdomen if the infection reaches the upper abdomen. An abdominal x-ray, upright and flat plate will confirm the presence of an ileus.

If on pelvic examination a mass is detected in the posterior or anterior vaginal compartments or above, the vaginal apex imaging studies should be obtained. Ultrasonography is helpful for the determination of pelvic masses, e.g., hematoma or abscess or free fluid collection. CT scan can also be of assistance in determining the exact location of the fluid collection, hematoma, or abscess. The presence of a hematoma in an infected patient should be considered as the site of infection, if present in the anterior or posterior vaginal compartment or in the pelvis. The radiologist can, frequently, differentiate a hematoma from an abscess. The postoperative patient, who develops fever, tachycardia, and elevated BC count and is found to have a hematoma at the vagina apex or in the anterior or posterior vaginal compartment, should be considered to have an infected hematoma. The surgical site is contaminated with the patient's own endogenous vaginal microflora and therefore, the infection should be considered to be polymicrobial.

Specimens should be obtained for the culture of facultative and obligate anaerobic bacteria. If a fluid collection (hematoma, abscess, free fluid) is present, the site should be aspirated and sent for gram staining as well as culture. The laboratory should be notified that a specimen is being sent and the site (abdominal incision, vaginal cuff, anterior or posterior vaginal wall) from where it was obtained. The gram stain results can be helpful in choosing the appropriate antibiotic therapy (Table 22.4). The gram stain can give suggestive information with regard to which bacteria may be present. If the there is a fetid odor to the fluid retrieved then consider the possible presence of anaerobic bacteria. Although *Staphylococcus* is a common cause of abdominal surgical site infection it is not a common cause of vaginal or pelvic infections.

The preliminary bacteriology report regarding the facultative bacteria should be available within 24 h, and between 24 and 48 h the bacterial identity and the antibiotic sensitivity pattern should be available. The results regarding anaerobic bacteria will take much longer, ≥72 h. Antibiotic therapy is

Table 22.4 Interpretation of gram stain results

Fluid characteristics	Gram stain	Tentative bacteriology
1. Serous (seroma)	WBCs rare	Sterile
2. Serous	WBCs 3+	Mycoplasma
3. Serous cloudy or purulent	WBCs 3+	
	Positive cocci in clumps	*Staphylococcus aureus*
	Positive cocci in chains	*Streptococcus agalactiae*
		Streptococcus pyogenes
	Positive cocci in chains +	*Staphylococcus* or *Streptococcus*
	Gram-negative rods	*Escherichia coli*
4. Frank purulence	As in #3	
Bloody	WBCs no bacteria	Hematoma
Bloody	WBCs + bacteria	
	As in #3	

empirically chosen, and therefore, the therapy initiated must provide broad spectrum antibacterial coverage. Infection that occurs within the first 24–48 h (early infection) is most likely due to facultative anaerobic bacteria, likely to involve both gram-positive and gram-negative bacteria. Whereas, infection that develops >48 h (late infection) is likely to involve both facultative and obligate anaerobic bacteria. Empirical antibiotic should begin with the simplest broad-spectrum coverage. In the absence of a pelvic mass, antibiotic can be added to the initial antimicrobial agent to increase the spectrum of activity, if the patient is not responding to the initial antibiotic therapy (Table 22.3).

Piperacillin/tazobactam has been shown to be very effective in treating postpartum endometritis, posthysterectomy pelvic infections, pelvic inflammatory disease, and pelvic abscesses.[44] The spectrum of activity of piperacillin/tazobactam is comparable to using the standard triple therapy, i.e., clindamycin + gentamicin + ampicillin; metronidazole can be substituted for clindamycin. The triple combination antibiotic therapy has been the so-called “gold standard” in obstetrics and gynecology. However, the broad-spectrum penicillins, such as, piperacillin/tazobactam, ticarcillin/clavulanic acid, and ampicillin/sulbactam offer the advantage of using a single agent to initiate antibiotic therapy if the infection is recognized early.[44–46] Ampicillin/sulbactam is not as effective as piperacillin/tazobactam in treating postoperative pelvic infections because of the decreased activity against *E. coli* of ampicillin/sulbactam.[47–49] Ampicillin/sulbactam should not be used as the sole agent in the treatment of postoperative pelvic infection because of the decrease in activity against *E. coli*. Patients not responding after receiving 48 h of therapy should be re-evaluated. The implication is that a resistant bacterium is present or there is an infected hematoma or abscess present.

Table 22.5 Antibiotics that can be used in place of aminoglycosides

Agent	Dose
Levofloxacin	500 q 24 h orally or intravenously
Ciprofloxacin	500 q 12 h orally or intravenously
Moxifloxacin	400 q 24 h orally or intravenously
Gatifloxacin	400 q 24 h orally or intravenously, if CrCl <40 mL/min the dose must be adjusted
Aztreonam (gram-negative facultative anaerobes)	1 g q 8 h intravenously (moderate infection) 2 g q 6–8 h intravenously (severe infection)

The patient who has failed initial antibiotic therapy may benefit from imaging studies, either Ultrasonography or CT scanning of the abdomen and pelvic with and without contrast material. If a patient who started on piperacillin/tazobactam is not responding within the first 48 h, consider adding gentamicin (5 mg/kg of body weight every 24 h, if the creatinine clearance is >80 mL min). The trough level of gentamicin should be obtained prior to the third dose and should be >2 μg/mL.[50,51] The administration of a β-lactam antibiotic, for example, piperacillin/tazobactam + gentamicin will provide synergy against Enterococci and streptococci. It should be pointed out that administration of a single dose of a cephalosporin for surgical prophylaxis could result in a sixfold increase in colonization by *E. faecalis*.[41] This should be taken into consideration when treating a patient for a postoperative pelvic infection who fails initial antibiotic therapy, if being treated with clindamycin or metronidazole + gentamicin. Alternatives to aminoglycosides are available but do not provide synergy with the penicillins (Table 22.5). These agents can be used in lieu of the aminoglycosides but are not really substitutes. The cephalosporins can be substituted for aminoglycosides and used in combination with clindamycin or metronidazole, but there is no coverage for Enterococci or methicillin-resistant staphylococci.

Patients who have a mass in the pelvis or anterior or posterior vaginal compartments can be treated initially with antibiotics. However, if there is no positive response within 48 h of initiating antibiotic therapy, drainage of the fluid must be performed. This procedure is best performed by taking the patient to the operating room, and under general anesthesia, the area can be adequately incised, explored, irrigated, and drained. It is best to use a closed drainage system under suction, e.g., Jackson Pratt or Blake drain attached to a suction device. The drains are usually left in place until the drainage is less than 30 mL over a 24 h period. The drainage fluid of any color other than serous should be considered abnormal. A specimen of the drainage fluid can be aspirated from the tubing and sent for Gram’s staining to determine if there are bacteria present. The specimen should be cultured for facultative and obligate anaerobic bacteria. If blood is exiting into the drain, serial hematocrits can be performed and a rise in hematocrit and volume is indicative of active bleeding.

The evolution from micro- to macroporous mesh has increased its use for repair of the anterior and posterior vaginal compartments with a reduced incidence of infection. There are four classifications of mesh (Table 22.6).[52] The macroporous prolene mesh typically used in pelvic reconstructive surgery is classified as Type I mesh. The two major concerns regarding the use of synthetic mesh in vaginal reconstructive surgery are erosion and infection. Infection of the monofilament prolene or monofilament polypropylene mesh can occur but the risk is relatively small. When comparing multifilament (silk, catgut, Dacron) with monofilament sutures, the former are associated with a significant potential for infection.[53] When comparing monofilament nylon to multifilament nylon suture, the infection rates were low and there was no significant difference between the two sutures with regard to incidence of infection.[54]Several studies have demonstrated that multifilament sutures have a higher risk of potential infection than do monofilament sutures, and monofilament polypropylene has the lowest risk of potentiating infection.[55–61]

Type I mesh appears to be the best suited for pelvic reconstructive surgery because it is a monofilament with a large pore size. The large pore size facilitates infiltration of macrophages allowing for bactericidal activity. The large pore size also allows migration of fibroblasts and infiltration of blood vessels, thus promoting host tissue growth.[62–64] A pore size in the mesh of <10 μm in each of their three dimensions is large enough for bacteria to migrate into the mesh, because bacteria are typically not larger than 1 μm, but does not allow macrophages and neutrophils which are too large to migrate into the pores to carry out phagocytosis.[65] Complications of mesh use in vaginal reconstructive surgery for pelvic organ prolapse are: (1) infection, (2) extrusion through the vaginal epithelium, (3) erosion into the bladder, urethra, rectum, and bowel, (4) retraction.

Infection in association with Type I mesh appears to be infrequent but does occur. There are basically two types of infection involving the surgical site; one in association with the mesh and the other is when the mesh becomes coated with a biofilm. Progression of the infection can lead to abscess formation or the development of necrotizing fasciitis. The difficulty is in differentiating extrusion of the mesh from infection associated with the mesh. In the case of extrusion of the mesh through the vaginal epithelium, extrusion typically occurs at the suture line or in an area where devascularization has occurred. The border of the extrusion site usually does not appear inflamed and there is no purulence or serous drainage at the site. The bacteriology, as one would expect, is typically polymicrobial and usually involves facultative and obligate anaerobic bacteria. Boulanger et al. found that polymicrobial infection was present in 31% of the cases and unimicrobial infection was present in 25% of the infections.[66] It has been the author's experience that with appropriate specimen collection, transport of the specimen in transport medium to support both facultative and anaerobic bacteria, and processing these infections tend to be polymicrobial. Infections developing within the first 24–48 h of surgery, however, will most likely be unimicrobial due to *Streptococcus agalactiae*, *Streptococcus pyogenes*, *E. coli,* or other gram-negative facultative anaerobic bacterium. The presenting signs and symptoms of cellulitis or abscess associated with mesh are similar to those in the absence of mesh. The presence of an abscess or biofilm formation on the graft requires surgical intervention. Not all abscesses require surgical intervention; if the abscess is located superficial to the mesh and under the vagina epithelium and not too distant from the suture line, spontaneous drainage and the administration of antibiotics may well result in resolution of the infection.

Table 22.6 Classification of synthetic mesh

Type I	Macroporous – pore size >75 μm. Polypropylene
Type II	Microporous – pore size <10 μm
Type III	Macroporous with multifilaments or microporous components
Type IV	Submicroporous mesh – pore size <1 μm (not used in pelvic reconstructive surgery)

Uncomplicated infection, cellulitis, can be treated with the administration of piperacillin/tazobactam 3.37 g IV every 6 h or Ertapenem 1 g IV every 24 h. However, if a polymicrobial infection is suspected then it would be best to administer a combination of antibiotics, e.g., clindamycin 900 mg or metronidazole 500 mg IV every 8 h + gentamicin 5 mg/kg of body eight IV every 24 h + ampicillin 2 g IV every 6 h. Some individuals will substitute ampicillin/sulbactam or piperacillin/tazobactam or ticarcillin/clavulanic acid for ampicillin. There is no need to remove the mesh when infection is detected early. The graft does not become infected initially, but can act as scaffold for the bacteria to adhere to and forms a gelatinous matrix known as a "biofilm." Bacteria are embedded within the biofilm, and more than one genus can become embed within the biofilm.

The biofilm is impervious to antibiotics, and macrophages and neutrophils cannot penetrate the biofilm. Therefore, the biofilm-coated mesh must be removed from the surgical site. Once the biofilm-coated mesh is removed, the bacteria embedded in the biofilm are also removed and the nidus of infection is removed. If there is any necrotic tissue present at the surgical site this tissue must be debrided. Once the wound has been evacuated and all necrotic tissue removed, antibiotic can enter the surgical site and functioning in conjunction with the patient's immune system, the infection can be resolved. Removal of the mesh that has become coated with a biofilm is relatively easy. Gently tugging on the graft is sufficient for the mesh to slide out of the surgical site. Unlike removing mesh from a noninfected field which is rather difficult to accomplish, the mesh often is removed in multiple pieces. Since infections occurring in one or more vaginal compartments and associated with biofilms tend to be polymicrobial, combination antibiotic therapy is suitable, e.g., clindamycin 900 mg every 8 h or metronidazole 500 mg every 8 h + gentamicin 5 mg/kg of body weight every 24 h + one of the penicillins every 6 h.

Summary

Pelvic reconstructive surgery should be considered as clean contaminated surgery, especially if conducted through and in the vaginal compartments. Administration of prophylactic antibiotics does reduce the risk for infection but does not eliminate infection. Patients with a *Lactobacillus*-dominant endogenous microflora are less likely to develop a postoperative pelvic infection than patients with an altered endogenous microflora, e.g., BV or a gram-negative facultative anaerobic bacterium that has assumed dominance. The presence of mesh in the operative site will enable infection, because it serves as a foreign body and will reduce the size of inoculum to initiate infection. Once the diagnosis of infection has been made, antibiotic therapy is initiated and continued until all of the patient's clinical parameters have returned to normal. The antibiotic regimen should be active against gram-positive and gram-negative facultative and obligate anaerobic bacteria. If there is an abscess or infected hematoma it will most likely require drainage. If the mesh has been covered with a biofilm it must be removed. Once the mesh has been removed, a new piece of mesh should not be installed.

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Rectal Complications of Mesh Repairs

23

Dennis Miller

Background

It is believed that rectal complications from gynecologic surgery are rare. A review of the current literature would support this assertion.[1] However, case series involving limited numbers of patients, often reported by referral centers, are likely to underestimate the actual number of complications. Even if rare, the inherent seriousness of rectal complications necessitates thoughtful consideration of their potential. If grafted repairs are associated with specific and distinct rectal complication, then consideration also needs to be given as to when the use of grafts is indicated.

There is no consensus as to whether the posterior vaginal compartment reconstruction requires graft reinforcement at all.[2] The issue is certainly more complex than simply reporting of rectocele recurrence rates with traditional plication. Posterior graft implantation is often used to prevent enterocele recurrence or as a more successful way to achieve apical support through the posterior compartment. These needs must be balanced against the complications discussed here.

It has been 10 years since DeLancey described the structural anatomy of the posterior compartment with histologic correlation.[3] There is a great distinction between the anatomy of the upper and lower posterior vagina and their relationship to the rectum (Fig. 23.1). The epithelium of the lower third of the vagina is thick and there is endopelvic aponeurotic tissue attaching the distal vagina to the pelvic side wall and perineal membrane preventing perineal descent and rectocele formation. There are decussating fibers of the levator ani, often referred to as the Pubovaginalis that reinforce the lower third of the vagina. It requires direct obstetrical damage or disruption of the levator ani to create a rectocele in the lower third of the vagina. By contrast, the upper two thirds of the posterior vagina are much thinner and are nearly completely absent of connective tissue above 4 cm from the hymen. If this portion of the posterior vagina is made vulnerable by changes in the posterior axial deviation of the vagina and enlargement of the levator hiatus, enterocele and high rectocele may develop. This lack of upper vaginal connective tissue posteriorly makes appropriate midline plication less gratifying and graft repairs more appealing. Even with Abdominal Sacrocolpopexy it has been shown that if the graft does not extend beyond the upper posterior vagina sequential posterior prolapse may occur.

Randomized trials have confirmed the superiority of transvaginal repairs over transanal procedures for rectocele repair, and the standard, familiar midline plication appears to have reasonable success without significant rectal complications.[4] However, this applies more readily to lower rectoceles than to high rectoceles and enteroceles. In addition, midline plication has been associated with high rates of dyspareunia especially when levator myorraphy is performed by less experienced surgeons. The addition of a graft may mitigate these risks but at a cost of potential rectal complications.

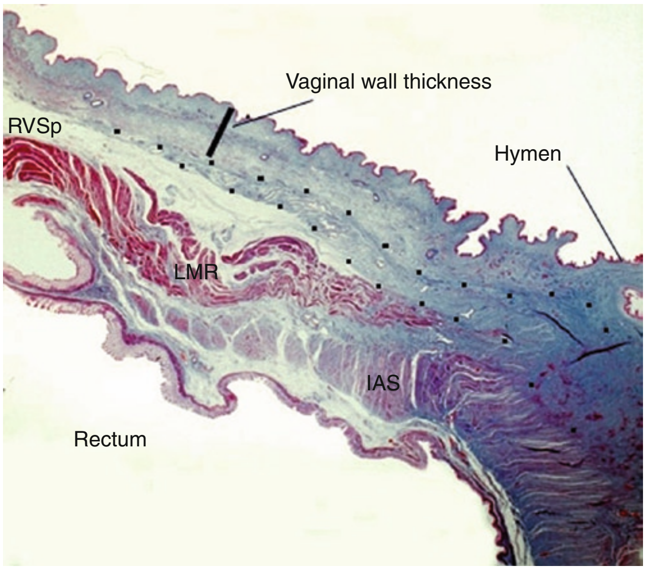

Fig. 23.1 Anatomy of the vagina and rectum (Reprinted from John DeLancey with Permission from Elsevier)

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Bowel Defecatory Dysfunction as a Complication

Regardless of the technique utilized, the goal of posterior compartment repairs has attempted to include correcting bowel evacuation complaints. Bowel defecatory dysfunction (BDD) is a poorly defined set of symptoms with variable description by patients. Three different categories of symptoms are reported. First is the need to digitally compress the vagina or perineum and occasionally digitally disimpact the rectum itself. In others, more sensory symptoms of incomplete emptying, post evacuation pressure, persistent need to defecate, and pain with defecation are reported. Finally, fecal incontinence may be associated with the pocketing of stool in association with a low rectocele. It is, of course, more likely to be associated with sphincter abnormalities. The Cochrane database review has concluded that the literature is incomplete in its ability to make distinctions about current surgical techniques' comparative ability to relieve these different symptoms.[4] This also confounds our ability to determine whether surgery can, at times, cause these symptoms as a complication.

Cundiff et al. have shown that patient satisfaction with posterior repair is more closely associated with relief of bowel defecatory symptoms.[5] The literature is conflicting as to whether posterior compartment repairs reliably alleviate bowel defecatory dysfunction.[6]

The challenge in repairing posterior compartment defects is that patient satisfaction does not correlate well with improvement in anatomic outcome.[6] It may be more technique dependent than in other pelvic floor compartments. If a procedure is able to maintain normal anorectal reflexes and the related sensory function of the distal rectum, it may be more successful in restoring and maintaining defecatory function. The question has been raised whether reducing rectal caliber and preventing perineal body descent are required to accomplish this goal. In addition, it is not known whether preoperative ano-rectal physiology studies can ignore outcomes and evaluate for the presence of other causes of BDD. We do know that the degree of prolapse is not well correlated with symptoms of BDD.[7] It would seem logical that other factors besides anatomy may play significant roles in optimizing outcomes.

It may be interesting to take patients with more severe symptoms and preoperatively assess them with defecography, transit studies, and colo-rectal consultation to determine whether persistent BDD symptoms can be minimized. Finally, even if posterior compartment repairs with grafts can be shown to improve defecation, we will need to show that they maintain durability of symptom relief in the face of long-term stress and Valsalva. In Altman's series there was a decline in success in regard to defecation after 12 months.[2] Grafted repairs add substance to the inherently deficient native tissue. In patients with levator disruption, we may find the best indication for graft reinforcement if we hope to have persistent durable results. Regardless, with achieved anatomic success, the symptoms that are most likely to remain alleviated are going to be the feeling of protrusion and need for digitation over the more multifactorial sensory-driven symptoms.

Obstructive Defecatory Symptoms

De novo obstructive bowel symptoms rarely occur following grafted repairs.[8] When it occurs, the pathogenesis is not completely clear. It is logical that if a grafted bilateral sacrospinous technique is used, that excessive tension on the graft may place the transverse margin of the graft across the rectum with potential resultant partial obstruction. There are times the graft margin is palpable on rectal exam. This can be verified on barium enema. However, palpating the margin of the graft may be normal and does not, in and of itself, demonstrate causality. The surgery itself may induce levator spasm and dyssynergia or uncover a preoperative predisposition to obstructed defecation. The immediate postoperative state is also confounded by the pharmacologic effects of narcotics and anesthetics on bowel motility. Mesh kits may actually help reduce the likelihood of tenting across the rectum in that the precut size of the mesh prevents the surgeon from custom cutting an excessively small transverse dimension. Most surgeons strongly recommend avoiding overtensioning the mesh over the rectum in trying to achieve the most aesthetic result, as this may lead to functional complications such as BDD.

Graft Erosion

The most feared rectal complication of posterior and apical graft repair is visceral erosion. There are only a small number of published reports to guide surgeons, and the most plentiful literature is surrounding the treatment of rectal prolapse with grafts.[8,9] Inference about factors that predispose to mesh erosion can be made from the data on vaginal erosions. A summary of possible contributors is listed in Table 23.1.

Table 23.1 Factors in rectal erosion

Material construction
Tension
Intraoperative placement
Depth of placement
Experience

We have learned a lot about material choice in the last decade. Meshes are now broadly categorized based on pore size and filament number. Low weight, monofilament polypropylene mesh with low flexural rigidity and reduced thickness currently appears to be least associated with erosion and other complications.[10] The reason manufacturers and surgeons have continued to report earlier errors in material choice and construction is that the mentioned characteristics of "safer" mesh may make them less palpably appealing and poorer in their handling characteristic.

Put another way, the best meshes are often the ones that are harder to work with. There are reports of Polytetrafluoroehstylene (Gore-tex and Teflon) erosion into the rectum at up to 7 years postop.[9] In one case utilizing Gore-Tex mesh for Abdominal Sacrocolpopexy, the entire 13 cm graft extruded through the rectum with spontaneous healing before any surgical intervention could be considered. This late, spontaneous extrusion without apparent infection or other pathology supports the notion that Gore Tex is a poor material choice (Fig. 23.2).

A second case similarly reported mesh being expelled per rectum with gentle traction followed by uncomplicated healing.[9] Transrectal removal of mesh with conservative treatment allowed complete recovery without rectovaginal fistula. These two cases are nonetheless reassuring with regard to conservative management of mesh erosion into the rectum.

Mersilene and Marlex meshes similarly were designed before materials science advanced to today's standards and is reflected in their higher exposure/erosion rate. Scientists have suggested that their morphological and mechanical properties may predispose them to increasing injury to surrounding tissues, including the rectum.[11] Within the more recent pelvic organ prolapse literature it is the use of Polyester mesh (another Type II multifilament mesh) used in the Infracoccygeal Slingplasty (IVS).[12–14] This type has had the most reports of adverse sequelae when used for posterior wall and apical repair. While the mechanism of adverse healing is still controversial, most experts would suggest that a multifilament mesh yields a greater inflammatory response and clinically higher rates of infection, biofilm coating development, and erosion. In one report by Baessler et al., 13 women were referred to a single center for pain and/or infection after Posterior IVS.[12] Another report revealed that 23.8% of patients getting posterior and apical application of a silicone-coated polyester mesh had major complications.[11] In addition to morphology, these meshes do not have the 20–35% elasticity which would match the compliance of the surrounding tissues.[10] Designing the ideal elasticity is a needed future goal, but we can hypothesize that inelastic meshes will disrupt the necessary compliance of the rectum for proper sensory function.

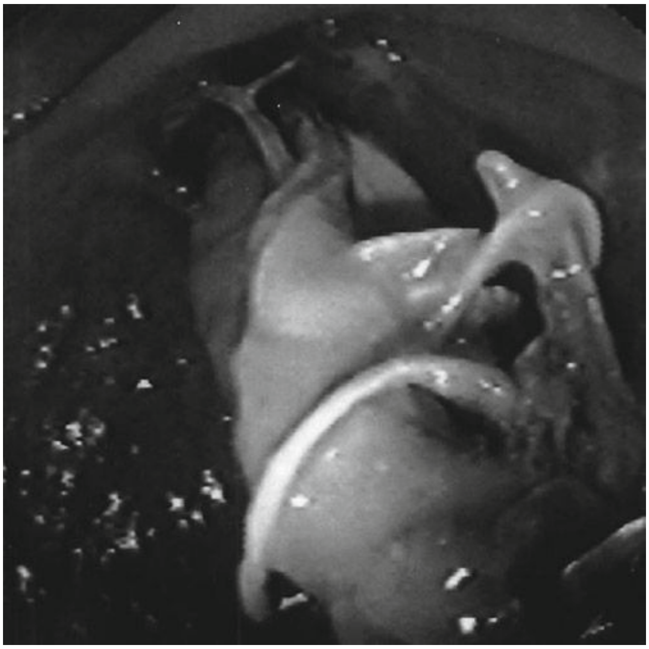

Fig. 23.2 Gore-Tex seen at colonoscopy (Reprinted from Kenton et al.[9] With permission from Elsevier)

Biologic grafts have thus far avoided reports of significant rectal complications. Many surgeons prefer their use to synthetic grafts in the posterior compartment, for exactly this reason. Unfortunately, the various heterogeneous materials available have produced inconsistent results and nonrectal complications. There is some scientific support for the theoretical advantage of a fenestrated, cross-linked or noncross-linked biologic graft at preventing the encapsulating thick layer seen in early biologics, perhaps due to the increased neovascularity possible or just the decrease in total material volume.[10] Xenografts are generally derived from porcine soft tissue due to availability, cost, and its high tensile strength. Nonetheless, not all biologics are created equally and choosing a porcine graft to theoretically reduce the already rare rectal complications without knowledge of its autolysis and subsequent failure rate is an uncertain trade off. In other disciplines, it has been seen that, as the collagen of the implant degrades, there is less enhanced endogenous collagen replacement than would be desired and that crystalloid calcification occurs.[2] This calcification has an unknown long-term effect on the rectum and potential late complications but requires as much scrutiny as the complication potential of a synthetic in the rectovaginal space.

Techniques to Avoid Erosion

It is tempting for the inexperienced surgeon to introduce tension during mesh application. The graft will appear more aesthetically pleasing when pulled straight and flat. Often, the tension is introduced when trying to avoid bunching or folding. While bunching is not desirable, it is important to avoid dorsal compressive force when placing the mesh. A harsh apical mesh margin may act as a "saw" predisposing to later rectal erosion. The appearance of industry-manufactured mesh kits may have had an adverse effect profile greater than the mesh itself. Mesh kits are designed to insert a graft in classic tension-free technique. The ability to place the mesh tension-free may be the single most important factor in avoiding rectal complications.

Rectal erosion of mesh is very uncommon and it is likely that when it does occur, technique has played a role. There may also be times that, what is seen as erosion, may more likely be surgical misadventure with occult placement of the mesh, partially or fully through the rectal wall. The space between the vaginal epithelium and rectal wall is thin and it is important to fully dissect the para-rectal space to be used for mesh placement and carefully deflect the rectum during passage of mesh fixation device. A balance is reached that can be learned only by experience. If the dissection is too shallow, vaginal exposure or stiffening is possible. If the dissection is too deep, inadvertent rectal injury is more likely.

Dissection of the posterior vaginal wall and the associated para-rectal space is very familiar to the legions of gynecologists performing posterior colporrhaphies over the last 90 years. However, a more educated technique may help to prevent rectal complications. While the rectovaginal tissue is thin, the initial dissection must be thick enough to leave a vascularized vaginal epithelium. Finding the balance between appropriate thickness and inadvertent entry into the rectum requires experience and following a few technical tips. Hydrodissection with an ample volume of fluid will find the path of least resistance and separate the layers. The full thickness of this layer has similarities to pastry and it is best to pass the syringe needle directly into the areolar space to get the best tissue separation. Avoiding blanching or development of an epithelial wheal helps confirm finding the right plane. Intraoperative rectal exam is inconvenient when the surgeon is trying to avoid contamination, but when done, clearly demonstrates the proximity of the rectum and helps the surgeon stay out of harms way. It is conceivable that the rectum could be entered without recognition. This can occur during the dissection.

Recently, more surgeons have been employing a distal transverse incision. It is often referred to in the colo-rectal literature as a transperineal entry. In posterior graft procedures, it may reduce incisional erosion but the reduced visibility may result in more potential for inadvertent proctotomy. With mesh kits that employ trocars, the blind passage of the needle provides a second location for rectal injury. Paraiso et al. placed trocars through the Ischiorectal fossa according to the directions for use and found significant proximity to the rectum during the most apical passage of the needle.[15] The other important finding was that these trocars may come in close proximity to the Inferior Rectal neurovascular bundle as it traverses the ischiorectal fossa. The only precaution during a tactile, blind passage is to be sure that the space is widely dissected and the rectum protected by the "receiving" hand. New generation kits attempt to solve this risk by avoiding the blind passage of trocars with interval fixation of mesh, but still necessitate the careful deflection of the rectum away from the fixation device.

Graft Procedures and the FDA

The FDA recently published a Public Health Notification regarding the use of Transvaginal Mesh (Table 23.2). There were over 1,000 reports of adverse events during 3 years of monitoring.[16]

Based on the years that were monitored, it is likely that the majority of reports was regarding slings for SUI and included adverse events that were related to the procedure but were unrelated to the mesh. It did not categorize the concern by compartment and so it is unknown how many of those reports were in regard to posterior compartment mesh. It is a challenge to assess the value of the report with such heterogeneous events included. It would be critical to know the incidence of occurrence for a given adverse event in order for surgeons to use the information in clinical practice. The other limitation is that there is no way to group the events by severity. The following is an FDA quote with regard to the nature of the problem. "The most frequent complications included erosion through vaginal epithelium, infection, pain,

Table 23.2 FDA Public Health Notification: serious complications associated with transvaginal mesh in repair of pelvic organ prolapse and stress urinary incontinence recommendations

Obtain specialized training for each mesh placement technique, and be aware of its risks
Be vigilant for potential adverse events from the mesh, especially erosion and infection
Watch for complications associated with the tools used in transvaginal placement, especially bowel, bladder, and blood vessel perforations.
Inform patients that implantation of surgical mesh is permanent and that some complications associated with the implanted mesh may require additional surgery that may or may not correct the complication.
Inform patients about the potential for serious complications and their effect on quality of life, including pain during sexual intercourse, scarring, and narrowing of the vaginal wall (in POP repair).
Provide patients with a written copy of the patient labeling from the surgical mesh manufacturer, if available.

urinary problems, and recurrence of prolapse and/or incontinence. There were also reports of bowel, bladder, and blood vessel perforation during insertion. In some cases, vaginal scarring and mesh erosion led to a significant decrease in patient quality of life due to discomfort and pain, including dyspareunia." One of the unintended consequences of the Public Health Notification is that it becomes potential legal fodder. It is inevitable that liability will play a role in the use of grafts in the posterior compartment, especially in this environment.

Despite of this, the conclusions and recommendations were in line with what most my thought leaders already believe. Grafted procedures, including those adjacent to the rectum, require experience and training. Achtari et al. found a significant impact of surgeon experience on vaginal mesh erosion.[17] This likely applies to the risk of rectal mesh erosion as well. There is a skill, involving dexterity and procedural facility, achieved over time, which reduces the number of adverse events. These procedures involve a greater level of understanding of anatomy and surgical principles. This mandates specialized training to achieve the best patient outcomes.

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Sexual Function After Mesh Repairs

24

Peter A. Castillo and G. Willy Davila

Introduction

It is estimated that 11% of the female population will undergo surgery for pelvic organ prolapse (POP) and/or urinary incontinence by the age of 85 years. Approximately 30% of these women will need reoperation for recurrent prolapse within 4 years of surgery.[1] These reported poor results have been associated with traditional approaches of anterior colporrhaphy, posterior colporrhaphy, combined anterior and posterior repair, as well as vaginal enterocele repairs. The recent evolution in prolapse surgery philosophy from a compensatory approach to a reconstructive or restorative surgery approach brings about a need for procedures with increased durability, and better efficacy.

The usage of implanted biomaterials for reconstructive pelvic surgery has become increasingly accepted following the widespread adoption of the tension-free vaginal tape (TVT) for the treatment of stress urinary incontinence. In fact, history will likely identify the TVT procedure as largely responsible for the recent development of needle and trocar-based kits using synthetic mesh for prolapse surgery.

The concept of using implanted materials for reinforcement or replacement of damaged or poor quality native tissue in the repair of pelvic organ prolapse follows along the same principles as used in general surgery for abdominal wall hernia repair.[2–4] This popularized analogy has also fueled much controversy regarding the appropriateness of synthetic mesh in the vagina, an organ with greater elasticity and less tissue bulk than the abdominal wall. Most recent publications have demonstrated a higher anatomic success rate for prolapse repair when a graft is used. However, functional assessments have not followed along the lines of anatomic results, with most studies showing fairly equivalent functional outcomes with native tissue and grafted repairs. Among the functional aspects impacted during prolapse surgery, sexual function is clearly one of the most important.

POP and urinary incontinence are strongly associated with reduced sexual arousal, infrequent orgasm, and dyspareunia.[5–8] Dyspareunia and sexual dysfunction following vaginal surgery for these conditions has been reported by various authors with conflicting results and will be discussed in this chapter. In our pursuit for improved, durable outcomes, it is imperative to consider both sexual activity and sexual satisfaction as outcome measures following surgical treatment of POP and incontinence.[9]

Definition of Normal Sexual Function

Nusbaum found that 66% of women aged 45–59 believe that a satisfying sexual relationship is important for maintaining a good quality of life (QOL) and that there are high rates of sexual concerns among women seeking routine gynecological care.[10] Additional studies have shown that female sexual dysfunction is a highly prevalent condition, affecting up to 40% of women in the USA.[11,12]

In order to understand and manage female sexual dysfunction (FSD), it is imperative to define normal sexual function in women. Previously, definitions of sexual dysfunction were based on human sexual response as defined by Masters and Johnson[13] and later revised by Kaplan[14] to include hypoactive sexual desire disorder. They describe a model of sexual response that assumes a linear progression from an initial awareness of sexual desire to one of arousal with a focus on genital swelling and lubrication, to orgasmic release and resolution. This linear progression of discrete phases has been challenged by various studies that describe overlapping phases of sexual response in a variable sequence that blends the responses of mind and body.[15] This overlapping sexual response cycle describes the importance of women being able to become subjectively aroused and that many psychological and biological factors may negatively influence this sexual arousability. Current DSM IV classification of FSD include: hypoactive sexual desire disorder, sexual aversion

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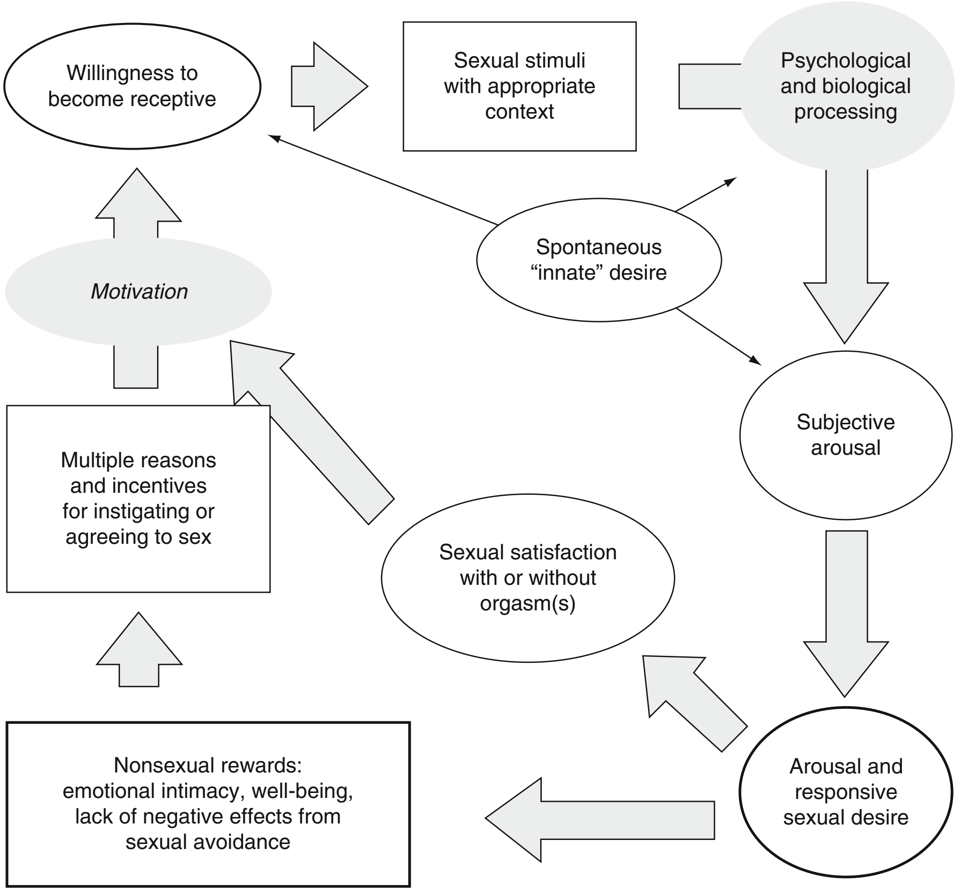
Willingness to become receptive
Sexual stimuli with appropriate context
Psychological and biological processing
Spontaneous "innate" desire
Motivation
Subjective arousal
Multiple reasons and incentives for instigating or agreeing to sex
Sexual satisfaction with or without orgasm(s)
Nonsexual rewards: emotional intimacy, well-being, lack of negative effects from sexual avoidance
Arousal and responsive sexual desire

Fig. 24.1 Female sexual response cycle

disorder, female sexual arousal disorder, female orgasmic disorder, dyspareunia, and vaginismus. The prevalence of dyspareunia is reported to be 8–21%[11] overall, and 15–21% of women ages 18–59. Causes of women's sexual dysfunction include interpersonal and contextual factors, personal psychological factors, and biological factors [16] (Fig. 24.1). At the center of this overlapping sexual response cycle lies sexual satisfaction, and it seems logical that vaginal surgery may affect sexual function and the sexual response cycle, either positively or negatively.

Dyspareunia Definitions

Under the DSM-IV FSD subcategory of sexual pain disorders, dyspareunia is defined as the recurrent or persistence of genital pain associated with sexual intercourse.[17] This is in contrast to vaginismus, which is the recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with vaginal penetration, which causes personal distress and noncoital sexual pain, is recurrent or persistent, and may be induced by noncoital sexual stimulation.

Assessment Instruments

Obtaining an accurate and clear sexual history from patients, especially those with sexual complaints, is frequently challenging and may require more than one visit for the patient to achieve enough comfort to discuss a sexual issue with her surgeon. Although there are a variety of QOL questionnaires for assessment of bladder and bowel function in women with pelvic floor dysfunction, there are only a few validated questionnaires to assess sexual function. The PISQ-12 is the most frequently used sexual QOL instrument for use in pelvic floor patients (Fig. 24.2). It has been validated in various languages and is now being modified to include an impact or bother scale.

Dyspareunia Related to Vaginal Surgery

Potential causes for sexual dysfunction after POP surgery include mesh erosion, change in vaginal dimensions, denervation/nerve damage, incontinence, pelvic floor muscle dysfunction, and factors that are partner-related or age-related.

Pelvic Organ Prolapse/Urinary Incontinence Sexual Function Questionnaire (PISQ-12)

Instructions: Following are a list of questions about you and your partner's sex life. All information is strictly confidential. Your confidential answers will be used only to help doctors understand what is important to patients about their sex lives. Please check the box that best answers the question for you. While answering the questions, consider your sexuality over the past six months. Thank you for your help.

1. How frequently do you feel sexual desire? This feeling may include wanting to have sex, planning to have sex, feeling frustrated due to lack of sex, etc.

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

2. Do you climax (have an orgasm) when having sexual intercourse with your partner?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

3. Do you feel sexually excited (turned on) when having sexual activity with your partner?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

4. How satisfied are you with the variety of sexual activities in you current sex life?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

5. Do you feel pain during sexual intercourse?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

6. Are you incontinent of urine (leak urine) with sexual activity?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

7. Does fear of incontinence (either stool or urine) restrict your sexual activity?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

8. Do you avoid sexual intercourse because of bulging in the vagina (either the bladder, rectum or vagina falling out?)?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

9. When you have sex with your partner, do you have negative emotional reactions such as fear, disgust, shame or guilt?

   ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

10. Does your partner have a problem with erections that affect your sexual activity?

    ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

11. Does your partner have a problem with premature ejaculations that affects your sexual activity?

    ☐ Always ☐ Usually ☐ Sometimes ☐ Seldom ☐ Never

12. Compared to orgasms you have had in the past, how intense are the orgasms you have had in the past six months?

☐ Much less intense ☐ Less intense ☐ Same intensity ☐ More intense ☐ Much more intense

Scoring:

Scores are calculated by totaling. The scores for each question with 0=never, 4=always. Reverse scoring is used for items 1,2,3 and 4. The short form questionnaire can be used with up to two missing responses. To handle missing values, the sum is calculated by multiplying the number of items by the mean of the answered items. If there are more than two missing responses, the short form no longer accurately predicts long form scores. Short form scores can only be reported a total or on an item basis. Although the short form reflects the content of the three factors in the long form, it is not possible to analyze data at the factor level. To compare long and short form scores, multiply the short form, it is not possible to analyze data at the factor level. To compare long and short form scores, multiply the short form score by 2.58 (12/31).

Fig. 24.2 Pelvic Organ Prolapse-Urinary Incontinence Sexual Function Questionnaire (PISQ-12) *Instructions:* Following is a list of questions about you and your partner's sex life. All information is strictly confidential. Your confidential answers will be used only to help doctors understand what is important to patients about their sex lives. Please check the box that best answers the question for you. While answering the questions, consider your sexuality during the past 6 months. Thank you for your help (Reprinted from Rogers et al.[18] With permission)

Table 24.1 Causes of postoperative pain and their suggested management

Category/site	Cause	Management
Atrophy	Dryness, loss of elasticity, and caliber	Local estrogen
Stricture	Reduced caliber, excessive plication, mucosa over-triming	Vaginal dilators Transection of band
Excess tone "Too tight"	Hypertonic levators	Biofeedback Smooth muscle relaxants
Apical pain	Reduced depth Focal pain	Vaginal dilators Trigger point injections (lidocaine, steroids)
Mesh-related:		
Erosion	Mucosal defect, discharge	Excision of exposed mesh
Contraction	Mesh "shrinkage"	Mesh arm – remove arm(s) Mesh body – remove central portion
Diffuse pain	Reaction to mesh, preexisting pain syndrome	Remove entire mesh Systemic therapy Avoid mesh use
Suture-related:		
Granulation	Reaction to (multifilament)	Remove suture

A careful and systematic pelvic exam is critical to identifying the site and severity of vaginal pain. The methodical exam must include an assessment of introital/vestibular pain, levator muscle tone, and any identified focal trigger points, urethral/trigonal/bladder pain, degree of mucosal atrophy, and identification of any strictures, erosions, stricture bands, or other abnormalities. Vaginal physical dimensions have been shown to not have any significant effect on sexual function. Actually, the site of vaginal surgery has been reported to have a more significant impact on postoperative dyspareunia rates.[19,20] (Table 24.1) As such, posterior vaginal wall repair has been associated with a higher rate of postoperative dyspareunia, especially if a levator muscle plication is performed.[21] In this situation, the pain may be multifactorial with pain related to reduced vaginal caliber and levator muscle spasticity.

Reported Experience Data

In general, when reviewing data on traditional or suture vaginal prolapse repair, there are significant discrepancies among studies with regard to sexual function postoperatively.

Several studies have demonstrated improvement in sexual life following pelvic reconstructive surgery due to resolution of incontinence or pelvic organ prolapse.[20,22–24] Additionally, some studies have shown that sexual function and frequency does not change following surgery. Following anal sphincteroplasty for fecal incontinence (FI), Pauls showed that FI of solid stool and symptoms of depression related to FI were correlated to a greater degree with poor sexual function. According to FSFI scores, they found that sexual activity and function scores after anal sphincteroplasty were similar to those of controls despite a higher severity of fecal incontinence in the former group.[25] In a separate study they reported that sexual frequency and function scores were unchanged after vaginal reconstructive surgery, with or without urinary anti-incontinence repair, despite anatomic and functional improvements.[26]

There is an inherent risk of developing dyspareunia following vaginal surgery – with dyspareunia occurring in as much as 20% of patients following plication of fascial tissue or levator muscles.[20] Additional studies have further supported this theory that sexual function may deteriorate following vaginal surgery, reporting as much as a 33% dyspareunia rate following posterior repair.[26–30] In fact, in a report on the effect of posterior colporrhaphy on sexual function, Kahn demonstrated an increase in sexual dysfunction relative to anatomic alterations along the posterior vaginal wall. Sexual function and dyspareunia rates have been noted to increase from 18% preoperatively to 27% postoperatively with posterior colporrhaphy.[21]

Sexual Function Following Grafted Prolapsed Repair

Improvement

Despite the current increase in graft use in reconstructive surgery, there is little published data to indicate the impact of vaginal mesh on sexual function in this population.

In 1 year follow-up after Apogee or Perigee with synthetic mesh for vaginal prolapse, Gauruder-Burmaster and colleagues reported a 93% success rate in terms of treatment of vaginal prolapse. When assessing sexual function in patients in this case series of recurrent prolapse, none of the 15 (12.5%) patients who reported preoperative dyspareunia reported the presence of dyspareunia postoperatively. This improvement of sexual function correlated highly with a high degree of patient satisfaction with a surgical procedure. Three percent of patients had mesh erosion, all of which were in the group of patients who underwent anterior mesh placement. No mesh infections were noted.[31] In separate analysis of this study population, the authors identified – using validated questionnaires – a significant number of non-urogynecologic, nonsurgical related factors, which affected sexual function in these patients.[32] They recommend that researchers should not jump to attribute a sexual problem to an operation without more in-depth evaluation of the patient's sexual function.

In a randomized comparison of anterior colporrhaphy versus anterior colporrhaphy reinforced with polypropylene mesh, focusing on sexual function, Nieminen demonstrated a significant reduction in palpable prolapse as well as reduction in dyspareunia scores in the mesh group. In this series, the mesh group had a prolapse recurrence rate of 11% compared to 41% in the traditional anterior colporrhaphy group. Mesh exposure was 8%. The authors concluded that the sensation of vaginal bulge was satisfactorily relieved with synthetic mesh implantation without resultant dyspareunia.[33]

In assessing the relationship between the Prolift system and dyspareunia, Lowman and colleagues assessed the rate of de novo dyspareunia in patients undergoing Prolift for vaginal prolapse. This study was performed via a self-administered questionnaire. De novo dyspareunia was reported in 17% of patients, with 75% of the patients reporting the dyspareunia to be mild to moderate. It was primarily introital dyspareunia. Interestingly, 83% of the patients who developed de novo dyspareunia would have the procedure done again due to the beneficial impact the mesh procedure had on their prolapse symptoms.[34]

Specifically assessing sexual function relative to mesh use in prolapse surgery, Nguyen and colleagues evaluated effect of standard anterior repair versus synthetic Perigee kit in women with advanced anterior wall prolapse. At 1 year follow-up in a randomized clinical trial, dyspareunia rates were noted to be 16% in the traditional anterior repair compared to 9% in the perigee group.[35] This study follows along other studies demonstrating that dyspareunia rates are not increased after mesh repair.

Worsening Function

Some studies have demonstrated deterioration of sexual function with the usage of synthetic mesh. Milani and colleagues reported on functional outcomes following anterior and posterior vaginal repair with Prolene mesh. In this series, the mesh was used as an overlay to fascial plication. The mesh utilized was hernia Prolene mesh that was available in 2003 and 2004. It was therefore not the lightweight mesh we are currently utilizing. In this series, the rate of sexual activity did not change but dyspareunia increased by 20%. This is despite an anatomical success rate of 94%. In women who underwent posterior repair with mesh, the dyspareunia rate increased in 62% of patients. Vaginal erosions of mesh were identified in 13% of anterior mesh cases and in 6.5% of posterior mesh cases. The authors of the study suggested that although there were good anatomical results with the use of Prolene mesh for prolapse repair, the significantly high morbidity rate, including dyspareunia, proved that this mesh was not appropriate for use in pelvic reconstruction. It must be kept in mind that the techniques used in this paper are not those currently utilized for mesh implantation and that the mesh material was of much higher weight than currently utilized.[36]

In an evaluation of sexual function after trocar-guided transvaginal mesh repair with Prolift mesh kit, Altman and colleagues determined that at 1 year follow-up overall sexual function scores worsened from 15.5 to 11.7 utilizing the PISQ-12 instrument. Interestingly, the overall worsening of sexual function was based on behavioral-emotive and partner-related items and not related to physical function. Overall, there was no deterioration in sexual function relative to changes in vaginal anatomy or usage of the mesh. This can also be seen as a lack of improved sexual function (based on PISQ-12 scores) with an anatomical cure of prolapse with synthetic mesh.[37]

Hamilton-Boyles and McCrery reviewed the presence of dyspareunia and mesh erosion after vaginal mesh placement with a kit procedure. They described the different etiologies for the occurrence of dyspareunia following mesh placement and attributed this condition to the presence of localized inflammatory reaction that could lead to myalgia. If this is the cause of dyspareunia, anti-inflammatory medications, local injections, and physical therapy may be used to manage the pain. If dyspareunia is secondary to excess mesh tensioning, mesh excision is likely needed. This goes along with other studies revealing that placement of the mesh under even mild tension, along with post-implantation mesh contraction, can lead to significant dyspareunia.[38]

Several studies have suggested that transvaginal mesh use does not significantly impair sexual function or change the number of patients who were sexually active.[24] According to some authors, however, patients should not necessarily expect an improvement in sexual function either.[39,40]

Various studies have evaluated the impact of usage of biologic grafts in prolapse surgery on sexual function. We found an improvement in sexual function, including a reduction in dyspareunia rates, based on PISQ-12 scores in women enrolled in a randomized, controlled study using bovine pericardium to reinforce midline fascial plication.[41]

Very few studies have compared biologic and synthetic mesh in prolapse surgery. In a recent randomized controlled trial comparing synthetic Gynemesh versus biologic Pelvicol for recurrent cystocele, the cure rate for cystocele was lower with the biologic graft, although it was associated with a higher erosion rate (6.3%). When evaluating sexual function, this group reported a more significant improvement in sexual function postoperatively in the biologic group ($p = 0.03$) as compared to the synthetic mesh group ($p = 0.31\%$). This may, in fact, relate to changes in mesh firmness after implantation.[42]

Surgical technique may have a role in the impact of synthetic mesh implantation on dyspareunia rates postoperatively. In a prospective trial of synthetic Prolift use in prolapse surgery with a continuous segment polypropylene mesh including an apical bridge, Milani and colleagues reported a 91% anatomic success rate with no increase in dyspareunia from pre- to postoperative evaluation (37%). Eighteen percent of patients had de novo dyspareunia and 28% of patients reported resolution of their dyspareunia. Thus, even when there is a mesh bridge along the apex of the vagina, there does not appear to be any significant increase in dyspareunia postoperatively.[43]

A recent review of a series of 17 cases in which mesh contraction was identified, revealed an increase in dyspareunia rate in these patients (100%). With the removal of contracted mesh arms and contracted mesh segments, dyspareunia decreased significantly in 64%.[44] Only three women required excision of the entire mesh.

Management of the Patient with Dyspareunia

As stated above, a methodical exam is key to identifying the site of vaginal pain. Once identified, treatment must be directed at the cause of the pain (Table 24.1). Local estrogen will likely benefit most patents, while removal of the entire implanted mesh is very rarely indicated – with the current use of only type 1 polypropylene mesh. Not infrequently, a combination of therapies is needed to normalize/improve postoperative sexual function. Importantly, the possibility of dyspareunia and other sexual dysfunction should be discussed openly during the preoperative informed consent process as there are patient satisfaction and potential legal implications.[45]

Summary

Due to the conflicting data on impact of mesh reinforced repair on sexual function, the benefits of durable results must be weighed against potential negative impact on sexual function. The onus of choosing the appropriate technique for pelvic reconstruction is on the informed pelvic surgeon and his/her patients.

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Part VII

Future

Reinforcement Materials in Soft Tissue Repair: Key Parameters Controlling Tolerance and Performance – Current and Future Trends in Mesh Development

25

Olivier Lefranc, Yves Bayon, Suzelei Montanari, Philippe Gravagna, and Michel Thérin

Introduction

Since their introduction in the late 1950s and early 1960s, we may expect that everything has been said, studied, and developed about mesh reinforcement in soft tissue repair. It is quite surprising though to see major market research institute forecasting Compound Annual Growth Rates (CAGR) of about 6% and 12%, respectively in Europe and the USA over the next 5 years (2008–2013).[1] That means that mesh uses should almost double in the USA in the next 5 years and increase by close to 50% in Europe. This overall growth in popularity is anticipated in almost all segments usually defined as soft tissue repair with a specific high trend in ventral hernia repairs and pelvic floor disorders. This growth is driven by a multitude of factors: aging population, obesity, expectation for better quality of life, increased demand for minimally invasive procedures, technological advancements, and extension of indication, awareness of the better outcomes provided by a tension-free repair.

Does that mean that the future is straight with the existing solutions? That would be presumptuous to think so. Men of the art would highlight some longlasting existing limiters: technologically advanced products will require more clinical support before being adopted; Also, demonstration of superiority will become more challenging for better outcome claims; and finally, reimbursement agencies will increase their expectation levels before granting clearance for new techniques and devices. Finally, some recent concerns published in the literature or highlighted by regulatory bodies about long-term chronic pain or tolerance issue associated with reinforcement materials[2–5] might slightly balance the enthusiasm generated by the tension-free concept in the last 20 years.

To better anticipate what should be the future trends in mesh developments, one might first analyze the sequence of events following the implantation of a reinforcement material in a host and what are the key properties of the meshes that will influence the host response in the proper direction.

The surgical procedure and the implantation of a foreign body will activate the healing cascade.[6] All the control mechanisms are not yet fully documented from a molecular biology perspective and interactions between the different players are complex. Hundreds of cytokines and messengers are involved in a sophisticated system.[7] However, the main events can be summarized as follows: the first week is dominated by the cellular inflammatory phase. In the absence of acute infection or immune reaction to the material which is the most frequent case, the macrophages are the key players, having the potential to switch the reaction toward the reconstructive phase where the fibroblasts and angiogenesis should reinforce the damaged tissues with a fibro-connective scar. The intensity and duration of the former determine the type and quality of the latest. The persistence of inflammatory stimuli will delay the reconstructive phase and usually intensify the scarring. The neocollagen formation is dependent on all the former events. Collagen type III is synthesized by fibroblasts within the first 10 days and then progressively turned in Collagen type I under the control of different enzymes and growth factors[8,9.] Fibroblast Growth Factor (FGF), transforming growth factor β (TGF β), metalloproteinases (MMPs), and tissue inhibiting MMPs (TIMP) are the most frequently evocated mediators of this phase.[10,11]

Figure 25.1 summarizes the cascade of events following meshes implantation. The objective of a well-designed material is to minimize the intensity and the duration of the

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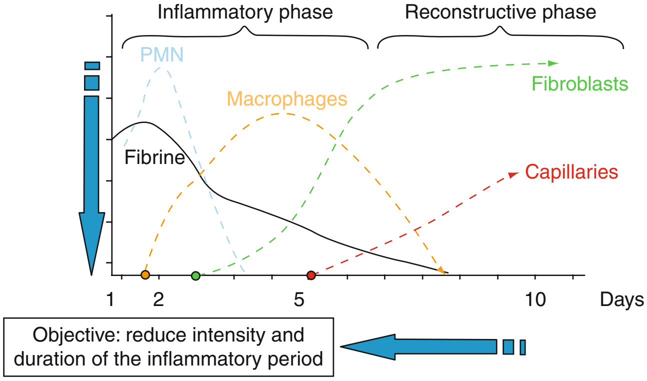


Fig. 25.1 Healing cascade following a biomaterial implantation

Table 25.1 Expected outcomes for an optimal repair following a mesh implantation. Relationship with the mesh properties

Expected outcomes for an optimal repair	The five key mesh properties
Limited scaring	Porosity
Quick integration/ Infection resistance	Surface properties
Comfort / compliance	Biomechanics adapted to recipient tissue
Durability	Stability
Tolerance toward adjacent viscera	Visceral adhesion prevention

inflammatory phase to rapidly support and restore the damaged tissues via a scar formation as close as possible to the native tissue.[12] Over the years, several mesh parameters have been identified as key drivers of the quality of the healing response. Table 25.1 summarizes the expected outcomes for an optimal repair following a mesh implantation and its relationship with the mesh properties.

The Key Role of Porosity in Scar Formation

It is today well established that the inflammatory reaction will have a major impact on the mesh behavior, contraction, and migration, after the implantation. The mesh contraction is directly linked to the inflammatory reaction and depends on the type and quality of the implanted foreign material.[13,14] As long as a mesh is recognized as a foreign material, inflammatory reaction will occur against it, up to generate fibrosis all around the filaments with a risk of capsule formation (Fig. 25.2).

The mesh raw material by itself has an impact on the mesh fibroblastic colonization and integration. However, for a same bulk material, the mesh pattern will also have a critical impact on the repair process outcome.[15] Among all physical parameters which characterize a textile, porosity seems to have the most considerable impact on the wound healing process, enhancing the tissue integration, with angiogenesis presence, fibroblast proliferation, and their collagen productions, while avoiding the mesh fibrotic encapsulation.[12,13,16,17]

In a knitted textile, one usually differentiates two types of porosity: the micro- and the macroporosity. The microporosity, in the micrometer range, is constituted of all small spaces existing between the fibers, monofilament or multifilament. When a yarn is made of a multitude of fibers such as in the so-called multifilament meshes, the microporosity dramatically increases. Such microporosity enhances intimate interface between the implanted material and the receiving host as long as the pores are big enough to authorize cell penetration.[18] This enlarged overall surface offered for colonization explain higher peeling strength from surrounding tissues obtained with a multifilament mesh compared to a monofilament mesh,[15], which could contribute to the early stabilization of the repair. Considering the size of colonizing cells, the pore size should be of a minimum of 10–20 μm. Below that range of size, there is a risk of bacterial penetration without getting access to the immune competent cells. The majority of knitted constructs exhibit such minimum pores sizes, which could not be obtained with tight braided (sutures type) or nonwoven (ePTFE type) constructs. The counter part of an enlarged implant surface is the risk to get more bacterial adhesion in case of massive contamination of the wound. This is the reason why, despite the long history of successful use of multifilament mesh in open abdominal wall repair[19,20,] the current trend is to use multifilament structure in clean cases (laparoscopic approaches, primary cases in inguinal repair…) and monofilament structure in more exposed procedures (pelvic floor repair through vaginal approach, multi-recurrent open cases with history of previous infection), consensus remaining on not implanting a foreign body material in case of existing clinical infections.[21]

The macroporosity, in the millimeter range, is constituted of all relative large pores existing between the columns of stitches. The knitting pattern controls the size, shape, and density of such large pores. The macroporosity has an even more important impact on the mesh integration because insufficient pore size will generate the mesh fibrotic encapsulation[16], which will bridge between the mesh yarns and then be responsible for the mesh shrinkage and potential pain or discomfort during tissue contraction. Figures 25.2 and 25.3 illustrate the encapsulation mechanism according different mesh pore sizes. From this study and others, it sounds that the cut-off in pore size to limit the risk of fibrous capsule formation and subsequent shrinkage is around 1–1.5 mm. Several teams showed that macroporosity was the key factor to control fibrosis.[14,22]

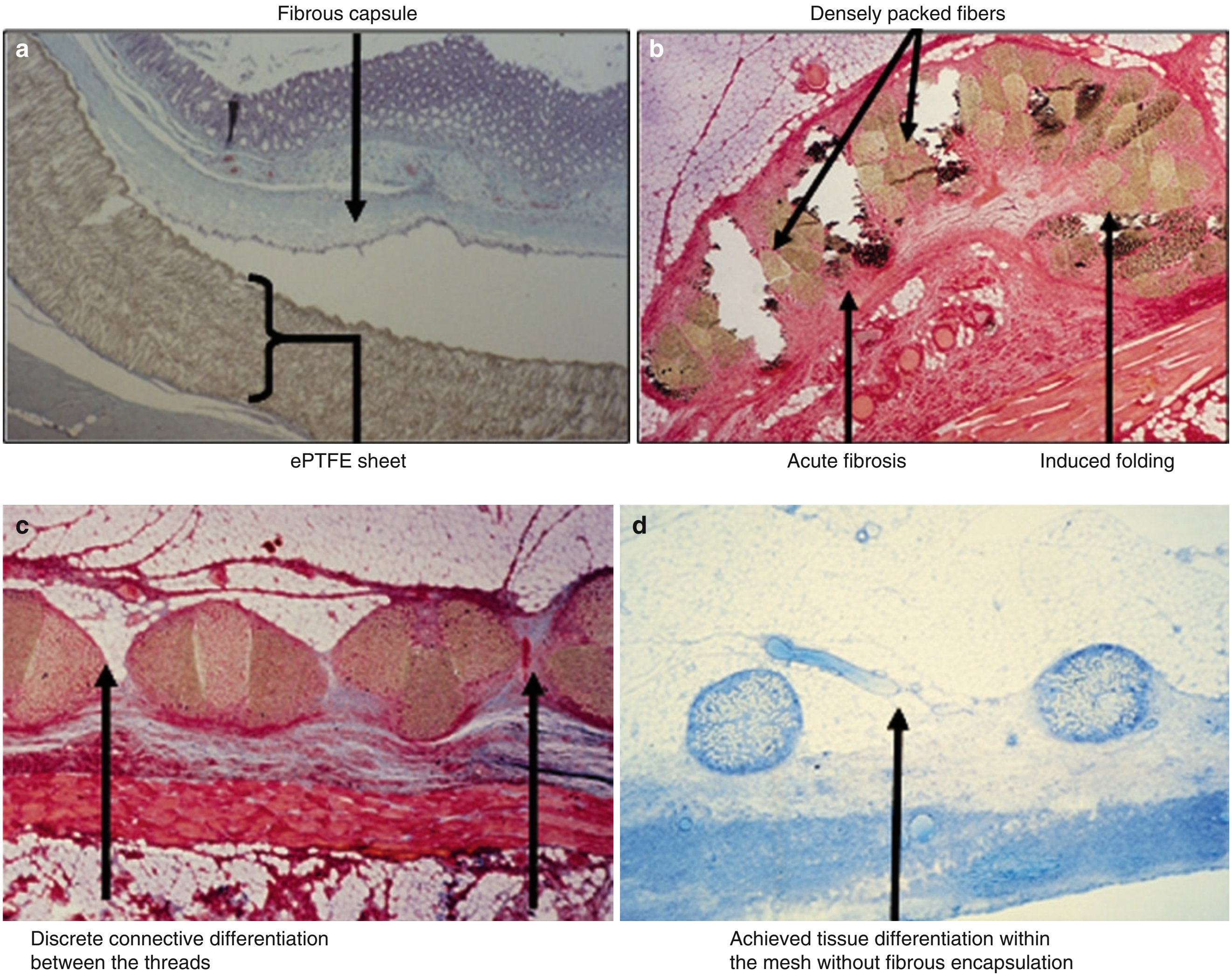

Fibrous capsule
Densely packed fibers
a
b
ePTFE sheet
Acute fibrosis
Induced folding
c
d
Discrete connective differentiation between the threads
Achieved tissue differentiation within the mesh without fibrous encapsulation

Fig. 25.2 Animal implantation study of different materials exhibiting various porosities: impact on fibrous reaction and intimate tissue differentiation. (HES or Masson trichrome, Obj ×5). (**a**) absence of cellular penetration within a dense material (ePTFE) with pores inferior to 10 µm. (**b**) Mesh material B (multifilament PET) exhibiting a microporosity larger than 10 µm but a macroporosity smaller than 500 µm. A thick capsule formation completely surrounds the implanted material to induce a folding. (**c**) Mesh material C (multifilament PET) exhibiting a microporosity larger than 10 µm but a macroporosity smaller than 1 mm Discrete connective differentiation (*left arrow*) but some fibrous bridges are still observed between some yarns (*right arrow*). (**d**) Mesh material D (multifilament PET) exhibiting a microporosity larger than 10 µm and a macroporosity around 1.5 mm. Absence of capsule formation. The *arrow* shows a neoformed loose connective tissue within the mesh identical to the one observed at distance

Macroporosity and microporosity have to be carefully separated from the mesh weight.[23] In the last 10 years, in an objective of simplification, large pores have been often associated to low weight, but these two properties are not always directly proportional and the weight by itself is not a key property to control the tissular reaction when the pore size is clearly a key parameter. Some authors have even shown that heavyweight large macroporous meshes could generate less adverse foreign body reaction than lightweight microporous meshes (Fig. 25.4).[24,25] In an attempt to describe a mesh which generates less fibrous reaction, the generic term of "large pore mesh" would be much more appropriate and less confusing than "low weight mesh."

If the mesh pore size is a key parameter, the pore geometry also has a critical impact on the foreign body reaction and tissue ingrowth.[26] When a mesh is implanted, it will sustain mechanical strain due to the tissue movement associated with the living host moves. The best mesh integration will occur for the meshes onto which the opened pattern remains under the physiologic host behavior.

An optimal mesh, in term of mesh integration and scar formation, would have to include all the parameters previously described. In particular, the mesh would have to associate a maximized porosity and adequate pore geometry, while providing the handling characteristics required by the practitioner and the procedure.

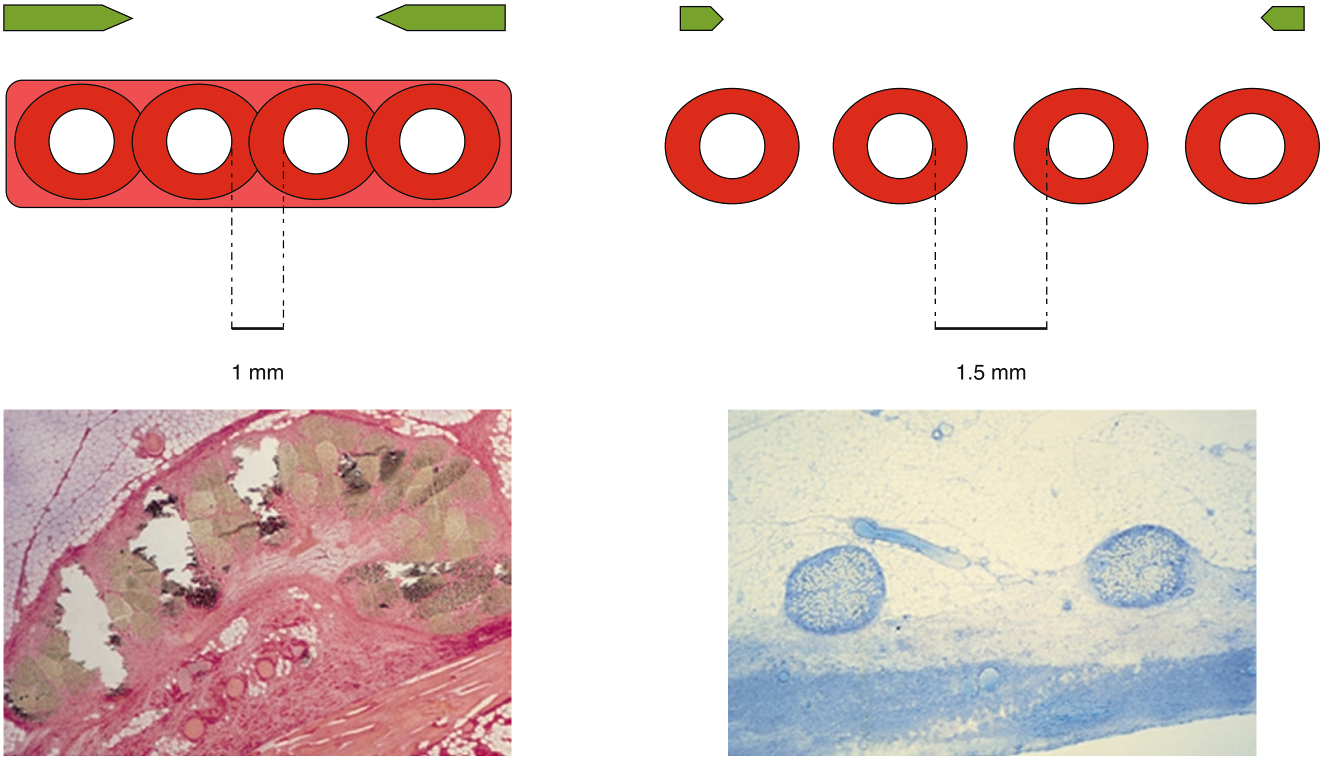
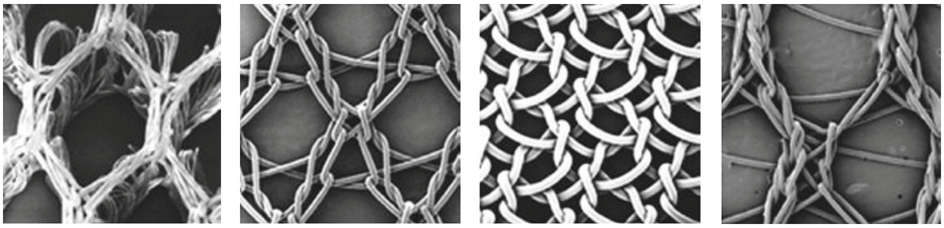


Fig. 25.3 Scheme summarizing how insufficient porosity increases the risk of shrinkage (HES or Masson trichrome, Obj ×5). Picture (**a**) shows a macroporosity inferior to 1 mm, with a resulting fibrous encapsulation and a lack of tissue integration in the textile construct. Picture (**b**) shows a 1.5 mm macroporosity. The textile threads are integrated by biological healing tissues while no encapsulation can be observed



Specifications	**PARIETEX$^{\text{TM}}$**	**PARIETENE$^{\text{IM}}$ light**	**MARLEX$^{\text{TM}}$**	**PROLENE$^{\text{TM}}$ Soft**
Textile type	Polyester multifilament	Polypropylene monofilament	Polypropylene monofilament	Polypropylene monofilament
Weight (g/m^2)	**78**	**38**	**90**	**41**
Thickness (mm)	**2**	**0.4**	**0.74**	**0.45**
Average porosity (%)	**97**	**89**	**87**	**88**
Pores size (mm) (h × l)	**2.55 × 1.73**	**1.5 × 1.7**	**0.84 × 1.04**	**1.7 × 2.5**

Fig. 25.4 Average porosity and pores sizes within different knitted meshes

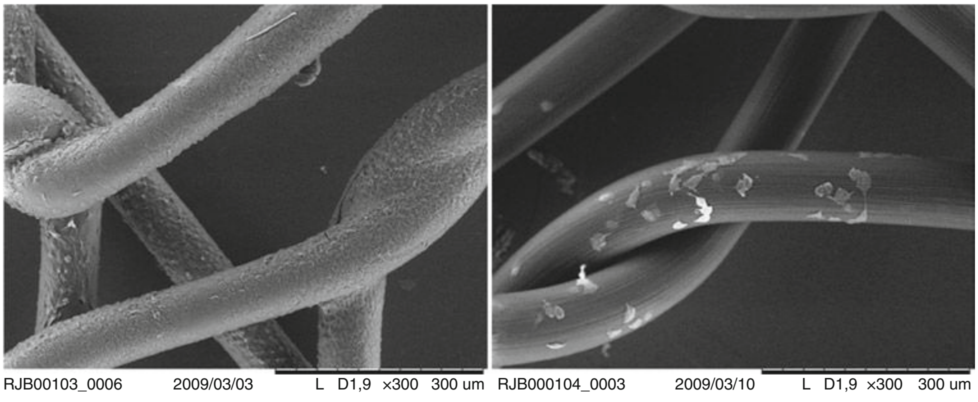
How the Surface Properties Influence Early Cellular Adhesion and May Influence the Competition Between the Host and Bacteria

As previously described, pore size and geometry can address partially the surgery outcomes; some physicochemical parameters of the surface have to be taken as well into account when considering mesh performance and optimization. The term "race for the surface" has been introduced decades ago and is still cited to describe the reaction, which occurs as soon as a medical device is implanted in a living host.[27] Microorganisms are dependent on substratum attachment for optimal growth and development. Implanted medical devices, with their artificial surfaces, tend to potentiate bacteria on their surface, so that normally nonpathogenic organisms become virulent pathogens. This feature is also enhanced due to the race for the surface occurring between microorganisms and host defense actors; the first reaction to establish a solid baseline will orientate the medical device integration. From a biological point of view, a strong link exists between wound healing and wound infection. Hence, wound infections, a major problem for all practicing surgeons, can be forms of acute wound healing failure. The risk of an acute wound infection is increased in the setting of an abnormal host inflammatory response.[11]

In order to increase the integration kinetic, which will improve the mesh integration ideally up to the point of native tissue and reduce the infection risk, the physical and chemical properties of the meshes's surfaces need to be carefully characterized and optimized.[17,26,28–30] For example, surface energy plays a critical role in the prokaryote and eukaryote cell adhesion and proliferation. The surface energy, or surface tension, also described as the surface wettability[31] , will define the surface hydrophilic or hydrophobic properties. This hydrophilicity can be evaluated by using physicochemical analyses, for example, contact angle measurement[31,32,] the low values (in degree) being relevant of hydrophilic surfaces while high values being relevant of hydrophobic ones.[33,34] As an illustration of this, polytetrafluoroethylene (PTFE) is one of the most hydrophobic polymer used as an implantable device with a contact angle of 105°[42], polypropylene (PP) is also classified within the hydrophobic category with a contact angle of 81°[33,34,] while Polyester (PET) is one of the most hydrophilic synthetic polymer used as a reinforcement material with a contact angle of 67°[33,34.] As a comparison, the natural polymeric surface for connective cells, the collagen, has a contact angle of 52°.[35]

Several authors in the literature have demonstrated that cell adhesion and proliferation is dependent on the surface hydrophilicity. 3T3 fibroblasts adhesion and proliferation have been shown to be enhanced with the surface hydrophilicity[36,] on surface chemical gradients ranging from highly hydrophobic plasma polymerized hexane (ppHex) to a more hydrophilic plasma polymerized allylamine (ppAAm) deposited on glass coverslips. Ren[37] confirmed these results on plasma modified silicone surfaces, which hydrophilicity was increased with allylamine, onto which dermal skin fibroblasts proliferated with an higher rate as compared with a nonmodified support. Sannino's group[34] extended these results on modified polytetrafluoroethylene (PTFE) supports, showing that surface energy and surface roughness both play a part on the adhesion process. As an illustration, Fig. 25.5 shows the difference in fibroblast adhesion between two meshes exhibiting the same knitting pattern but made of two different materials: polypropylene on one hand, and polyester on the other hand. In this experiment, polyester significantly authorizes earlier cellular adhesion/proliferation than polypropylene.

On the other hand, highly hydrophobic plasma-polymerized diethylene-glycol-dimethyl-ether surfaces prevented fibroblast adhesion, showing that cell adhesion can be either enhanced or inhibited by the surface energy.[38] This phenomena was also reported on highly hydrophobic fluorocarbon surfaces, whereas adhesion of *Staphylococcus aureus* was supported[39,] providing evidence that hydrophobic surfaces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PET progrip

PP progrip

Fig. 25.5 SEM images of PET and PP monofilament Progrip™ meshes seeded with L929 fibroblasts after 6 days of incubation. The meshes were seeded with 300,000 cell/mesh under axial rotation for 2 h and transferred in a 24 wells cell culture plate for incubation (37°C, 5% CO_2). After 6 days of incubation, the seeded meshes were fixed in methanol and observed under a degraded vacuum mode of an Hitachi TM-1,000 SEM

could show selective bioactivity, supporting the attachment of a microbial pathogen while decreasing the adhesion of host defenses eukaryote cells.

Several authors[40,41] showed that an active antibacterial coating can be combined with an increased hydrophilicity. For example, Quaternary ammonium plasma deposition on polymeric surfaces presented antibacterial properties against gram-positive and gram-negative bacteria while enhancing the surface wettability for an enhanced cell adhesion.[41]

In conclusion, there is a high probability to see in the future the surface of reinforcements materials moving from passive hydrophobic as polypropylene provides today to smarter surface combining increased hydrophilicity and antimicrobial activity for an improved tolerance.

Soft Tissue Biomechanics: Are Mechanical Properties of Meshes Adapted to All Kinds of Receiving Sites?

For several decades, the most frequent question asked about mechanical performance of reinforcement materials was: are meshes strong enough? After millions of implantation in different indications of soft tissue repair on one hand and published or regulatory declared reports of true mechanical failure of less than one per ten thousand on the other hand, it appears appropriate to conclude that the ultimate tensile strength is not really an issue of clinical relevance. Even with the substantial decrease in mesh density observed over the last 10 years, and the subsequent decline of the mechanical strength of such materials, most of the publications[30,42,43] agree that the level of safety in terms of ultimate tensile strength is still sufficient. Does that mean that the literature is clear in terms of what should be the minimum properties per indication and technique: not at all. In fact, there has been limited attention to investigate the mechanical characteristics of soft tissues.[43–47] In 2005, Cobb[48] measured the abdominal pressure during different maneuvers. The higher pressures were recorded for standing cough and jumping. This maximum pressure was in the range of 20–30 kPa. Using the law of Laplace and simplifying the abdomen to a cylinder of 30–35 cm of diameter, this max pressure corresponds to a maximum superficial tension supported by the abdominal wall or pelvic floor of approximately 20 N/cm. This could be, in theory, the maximum tension supported by a mesh assuming a worst case scenario of full replacement of either the abdominal wall or the pelvic floor. Other authors[43] established that limit at 16 N/cm using a similar approach. This would need further investigations and validations; however, most, if not all, meshes available on the market are significantly over those numbers.

The capacity of elongation of reinforcement materials under physiological loads appear to be a more critical parameter as the stiffening effect provided by meshes has been reported to induce discomfort and lack of mobility in reconstructed abdominal walls[26,47] or dyspareunia in pelvic floor repair[49.] The fibrosis/shrinkage, which may result from the healing phase (see corresponding paragraph), is expected to emphasize that effect.

Figure 25.6 presents the strain versus strength curves of various soft tissues and meshes. From this figure and assuming that porcine tissues are analogous to human, we can say that fascia and aponeurosis are two times stiffer but in the mean time weaker than muscles. Muscles are able to support massive deformation before getting torn (>100%). The strain versus strength curve shows a relatively linear behavior for aponeurosis while the curve for muscle exhibits a viscoelastic

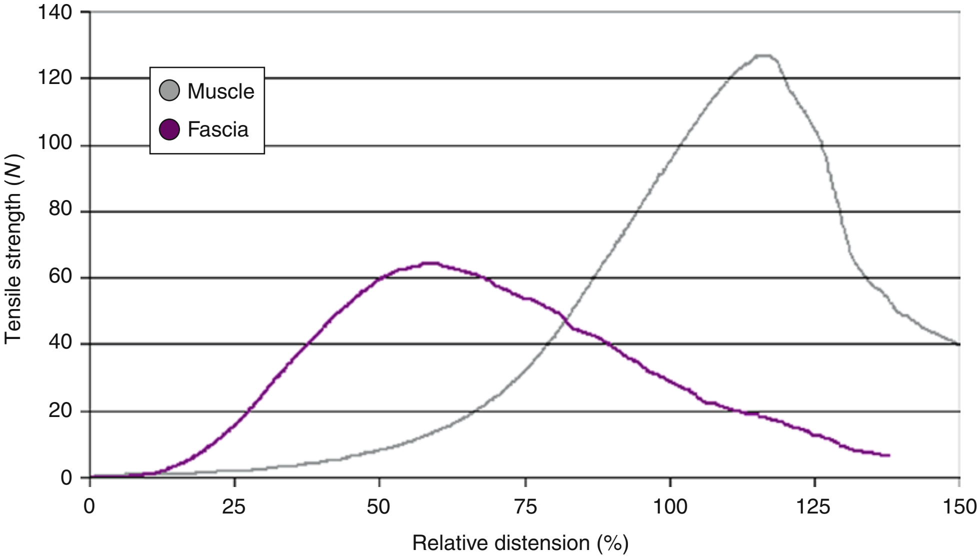

Fig. 25.6 Relative distension of a pig abdominal wall

behavior. In other words, in complex composite anatomical structures associating different layers of muscles and aponeurosis such as the abdominal wall, the weakest point is the aponeurotic or fascia component. That is the reason why most of the hernias occur in areas where muscles cannot backup any fascia deficiencies such as the *linea alba*, the inguinal region or former incision. When speaking specifically about pelvic floor disorders, the vagina plays a critical role as most of the prolapses occur through the vaginal wall. Its complex histological structure made of smooth muscles and extra cellular matrix rich in collagen and elastin gives the vagina unique properties. Some recent publication[50] evaluated the longitudinal mechanical properties of the vaginal wall from patients suffering from prolapses or from fresh cadavers without prolapse. The obtained curves show a behavior of visco-superelastic material with massive capacity of elongation under relatively modest stress and high variations from patient to patient.

When comparing the elongation of different meshes under physiological loads, Fig. 25.7 shows significant differences pending the knitting pattern and type of yarn. When modern meshes could match pretty well the mechanical behavior of fascia, small pore's old generation meshes made of large diameter monofilament are usually stiffer than any anatomical structure. That means that such meshes will impose their mechanical behavior on the anatomical structure it intends to reinforce. The risk of lack of compliance and subsequently the risk of discomfort is theoretically increased. This was confirmed in ventral hernia repair where it has been shown that stiff material induced discomfort and pain.[47]

The differences of behaviors between anatomical structures and sites mean that the reinforcement material would benefit from being specialized to a given site and indication, assuming that the fundamental understanding of what should be the ideal properties exists.

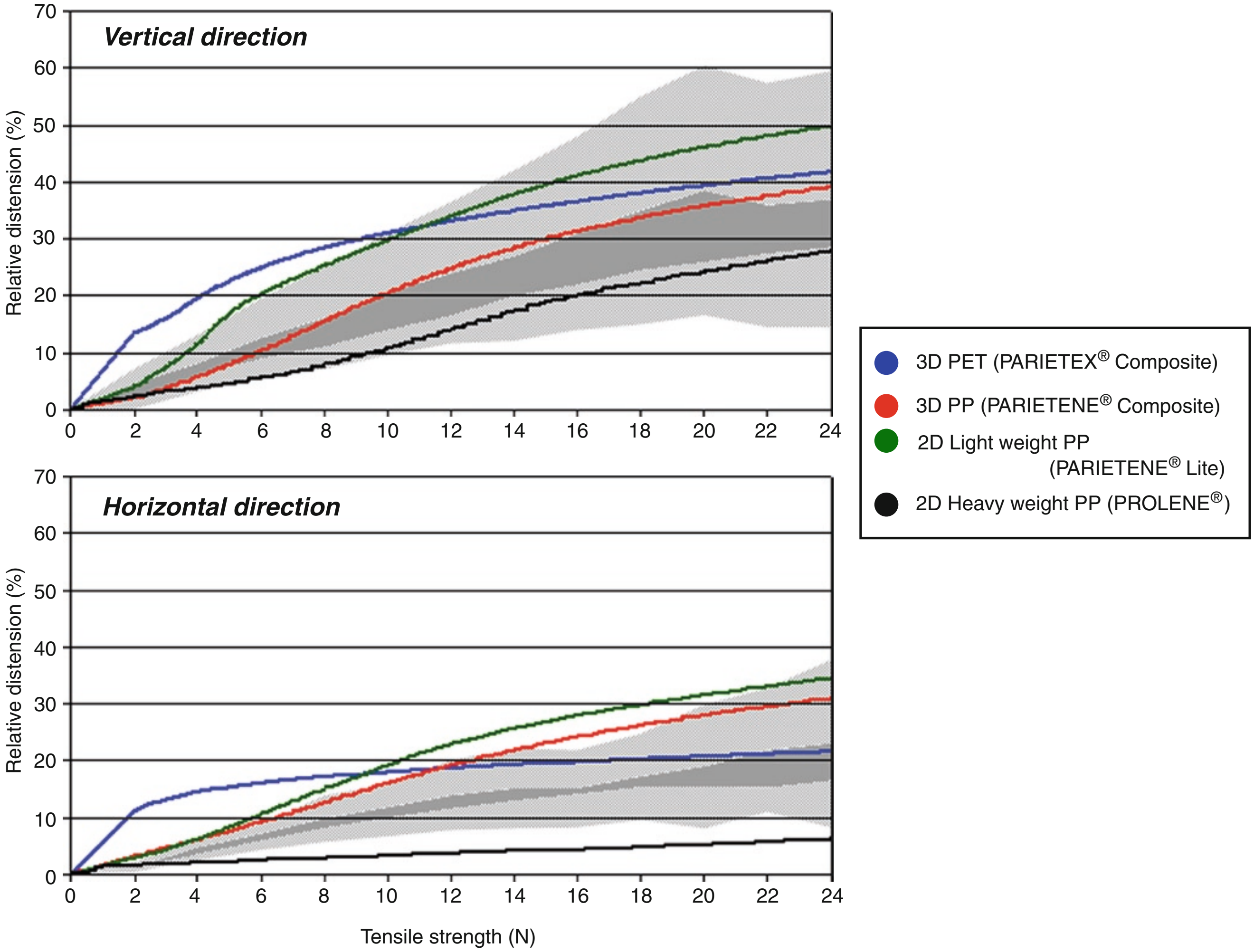

Fig. 25.7 Relative distension of the human abdominal wall versus elasticity of various meshes

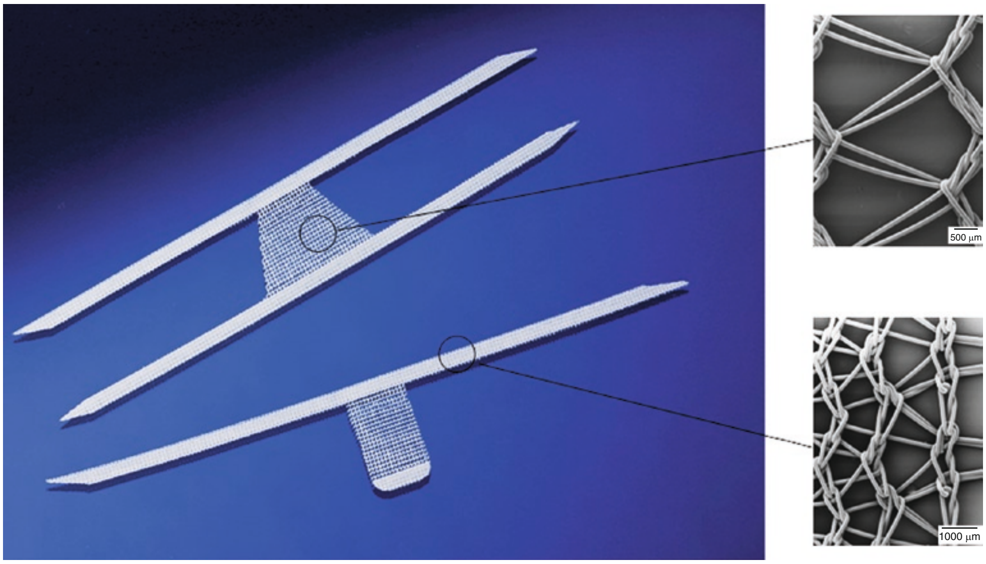

Fig. 25.8 Quadra and duo meshes. Mesh structure is represented on SEM micrographies

Figure 25.8 presents two meshes specifically designed for pelvic floor reinforcement through vaginal approach. One may note lateral slings made of a different knit than the central piece. The lateral slings are intended to provide the anchoring effect while the central piece is intended to provide the suspension. In such intent, the lateral slings are stiffer than the central piece.

Are Synthetic Materials Stable Enough to Provide Long-Term Guaranty of Durability?

Polypropylene (PP) is the most commonly used polymer in mesh design and has a strong reputation of inert material.[51–53] However, recent studies[53–55] have indicated that the knowledge concerning long living host interactions with this polymer is still limited and that this material may not be as inert as commonly believed. Bracco[54] has studied 21 excised polypropylene hernia meshes after an average period of implantation of 32.5 months and showed that these meshes have sustained morphological degradation. SEM images revealed cracks and fissures on the filament surfaces. These characteristic cracks of polypropylene yarns from excised hernia meshes were also found in two different studies from Costello.[53,55] The origin and mechanism of this degradation is not yet clear and some controversies between the authors can be noted. While Costello affirms that the observed damages are caused by oxidative degradation, Bracco suggests that the absorption of small organic hydrophobic molecules by the hydrophobic mesh filaments could be at the origin of the observed damages. Interestingly, these results are consistent with other observations made on suture materials in ophthalmological applications, where some authors[56–60] have reported morphological degradation of polypropylene surface in human eye.

Polyethylene terephthalate (PET), also known as Polyester, has also been commonly used in soft tissue repair meshes for almost 50 years. Contrary to some suspicions related to hydrolytical degradation of PET filaments,[61] no damage was noted in the excised PET samples observed by Bracco.[54]

Recently, Clave and collaborators[62,63] have studied 84 excised samples used in Pelvic Floor Disorders. The meshes were either made of PP (71 samples) or PET (13 samples). From the excised samples 39% of polypropylene meshes showed some surface degradation while no damage was noticed on polyester-based materials (Fig. 25.9). According to Clave's data the PP degradation seems to be significantly correlated to infection or inflammation as histologically reported. This observation could indicate that in vivo PP degradation might be related to adverse host reactions, which need to be more precisely investigated (chronic nonseptic inflammatory reaction, subclinical infection…).

The clinical relevance of such degradation findings is still unclear. Is it a significant contributor to some complications such as dyspareunia, abdominal chronic pain or just an anecdotic consequence of the long-term presence of a foreign body in an adverse inflammatory environment? Is it a vicious circle where degradation continuously stimulates inflammation which even further degrades the material as observed with wear debris and bone resorption in joint replacement? Future investigations are needed to clarify the importance of this phenomenon. If confirmed at a large scale, those polypropylene degradations may reinforce the interest in other polymers such as polyester or other polyefines for future mesh development.

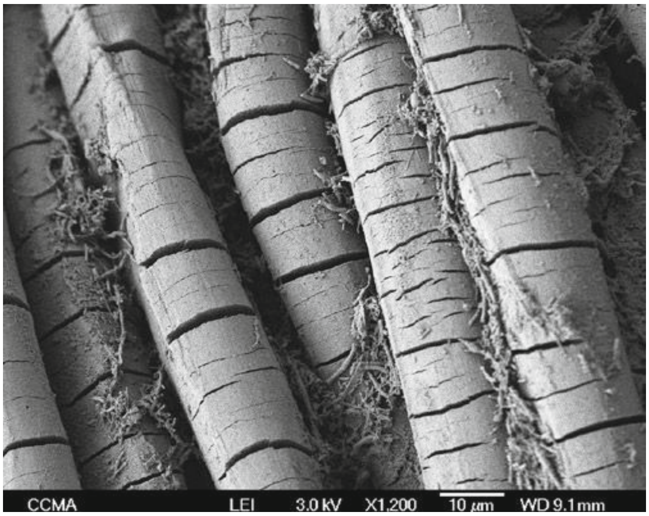

Fig. 25.9 SEM observation of degraded PP mesh under septic environment

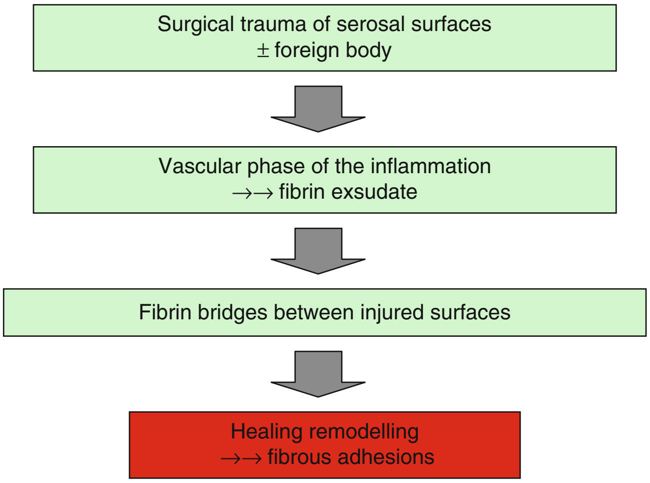
Surgical trauma of serosal surfaces ± foreign body
Vascular phase of the inflammation →→ fibrin exsudate
Fibrin bridges between injured surfaces
Healing remodelling →→ fibrous adhesions

Fig. 25.10 Schematic cascade of events driving the formation of fibrous adhesion

How to Combine Rapid Tissue Integration in Soft Tissue Repair and Protect Hollow Viscera at the Same Time?

Adhesions are abnormal attachments between tissues or organs that form after an inflammatory stimulus, most commonly surgery. When adhesions affect normal tissue function, they are considered as complication of surgery.

Adhesion formation between pelvic structures, secondary to surgery or pelvic disease, is a significant cause of infertility.[64] Some surgical procedures are particularly sensitive to adhesion formation; examples include procedures near tubular organs, such as fallopian tubes or the small intestine, procedures on uterus, such as myomectomy, where the formed adhesions can constrict and thus obstruct the organs.

The cascade of events (Fig. 25.10) that drives the formation of fibrous adhesion is now well documented. The erosion or trauma of the serosal surface lead to inflammatory exudation or bleeding and provides a source of fibrinogen, which after deposition on two adjacent surfaces polymerize into fibrin through the enzymatic action of thrombin. Otherwise unattached both surfaces consequently adhere to each other. The neoformed fibrinous adhesions may then persist because damage to the cell environment compromises the normal fibrinolytic activity. If fibrin is not removed, the temporary fibrinous adhesions will then develop into permanent fibrous adhesions.[65] Macrophages, blood vessels, and fibroblasts invade the fibrinous meshwork under the influence of growth factors, and inflammatory mediators. Collagen and other connective tissue elements are laid down to form the permanent band-like adhesions.[66] The presence of a porous mesh potentiates this cascade.

Understanding this sequence, it then seems logical to propose the use of appropriate medical treatment or surgical devices to prevent or minimize the adhesion formation.

There are two major approaches:

- Minimizing surgical trauma
- Use of barriers to prevent adhesions

Any action that limit surgical trauma will in theory reduce the adhesion formation: talc free gloves, less reactive suture or mesh material, avoidance of bleeding, minimal invasive surgery. Dr Ray[67] raised the point that the increased use of laparoscopy for abdominal procedures between 1988 and 1994 did not induce a massive concomitant reduction of hospitalization rate for bowel obstruction, suggesting that minimal invasive surgery still request adhesion prevention means.

Barrier devices used for the prevention of postoperative adhesion formation include both films and gels. In principle, the physical barrier, film or gel, is interposed between two surfaces preventing their adherence by the fibrinous bridge. Once remesothelialization has occurred, the physiological barrier is reformed preventing adhesion on the long term; then the implanted barrier is no longer needed. With peritoneal tissue, the complete mesothelial repair lasts 8 days[68]. New mesothelium develops predominately from islands of epithelial cells that attach throughout the wound from surrounding visceral peritoneum, then proliferate and expand the wound coverage. This explains that large defects heal about as quickly as small ones.[68] One can understand from the mechanism and environment of adhesion formation that ideally the barrier should not interfere with wound healing, remain efficient in the presence of blood, be continuous, be compliant with organ shape and mobility, be bioresorbable, and be usable in open and laparoscopic surgery. The barrier effect can be combined with a reinforcement material in one single device. The risk associated to adhesion formation is much higher when a foreign material is implanted in intraperitoneal situation or in close contact with hollow viscera. It then becomes essential to protect the device from contact with fragile organs within the first 7 days following the implantation. Several studies have recently compared the performance of such composite devices in term of adhesion prevention and tissue integration[69–71] demonstrating that superior performance is accomplished with bioresorbable continuous hydrogel film associated with mesh compared to nonresorbable barrier such as PTFE.

The availability of numerous technologies to address the clinical outcomes of adhesion formation provides substantial progress in the management of postsurgical adhesion prevention. Yet, important questions remain: Why do some patients form adhesions to trauma which do not lead to adhesions in other patients? Why do some adhesions form distant to the trauma area? What is the relation between adhesion formation and pain? How important is it to protect hollow viscera outside the peritoneal cavity? Does adhesion prevention barrier prevent erosion in vaginal surgery?

For the two latter questions, recent literature[72–74] tends to support that even in the retroperitoneal space hydrogel barrier seems to have an interest in the reduction of erosion rate after mesh implantation through vaginal surgery. However, the experience in vaginal surgery of such composite products is still limited and has not yet reached the level of evidence which has been accumulated over the last 10 years of use in the peritoneal cavity.[75] This is definitely a domain where significant improvement is expected in the future as mesh tolerance to surrounding hollow organs remains a concern in pelvic floor repair especially through vaginal route. The potentially contaminated environment, the frequency of significant bleeding and/or associated gestures such as hysterectomy, colpectomy and various plications, the proximity of the mesh from the wound incision, the narrow space which make difficult the correct spreading of the material are factors that may contribute to increase the risk of adhesions and erosions and might balance some of the benefit of the tension-free repair provided by meshes.

Is There a Room for Regenerative Medicine and Tissue Engineering in Soft Tissue Repair?

Despite significant improvements of synthetic meshes, and all the ways of further promising enhancements described before, there will still be occasions when this approach will not provide ideal results. There are a number of limitations that stem from the fact that a polymeric mesh will remain synthetic and therefore will not have the ability to physiologically replace living tissues and functions. Such situations may be encountered in complex abdominal wall reconstructions with significant domain loss, severe prolapse in elderly patients, multirecurrent infected hernias, etc…

Biologic meshes, xenogenic or allogenic grafts mostly obtained from dermis or small intestine submucosa[76–78] have been developed and introduced in order to provide an alternative solution in such complex situation. The biologic meshes, due to their collagen composition and native architecture, are expected to not induce the same foreign body response as the synthetic meshes do. By providing a physiological substrate to the host cells and limiting the nonspecific inflammatory reaction, these materials have the theoretical property of being progressively remodeled.[79] The balance between remodeling and neotissue formation is critical to maintain performance over time. The quality of the purification and an adequate stabilization of the collagen structure are the two key criteria that control the balance and overall tolerance of these materials. When these parameters are adequately controlled, promising results have been published even in severe cases.[79–81] This explains their recent increase in popularity especially in the USA.[82]

This could be considered as a first step in the direction of regenerative medicine when the implant is progressively incorporated and then replaced by physiological tissues. In this first approach, the regeneration relies on the host capacity to self-recruit the adequate cells and to induce their differentiation by autologous means in the expected path. One could envision that this “passive” path could be, in a second step, replaced by an “active” induction by incorporating biological growth factors in the scaffolds. This approach has already provided successful results in orthopedic applications where significant bone induction has been obtained by incorporating recombinant Bone Morphogenic Protein (rBMP) in a collagen sponge.[83] The ultimate step would be to develop in vitro a hybrid construct combining living cells and scaffolds. This approach called Tissue Engineering (TE) appears technically feasible but has not yet reached the industrial scale-up phase for surgical applications. All these active approaches are extremely promising for replacing tissues exhibiting poor spontaneous regenerative capabilities such as, for example, neural, cardiac, and cartilage tissues. However, one may argue that the involvement of such highly differentiated tissues is limited in soft tissue deficiencies and consequently the benefit of such sophisticated technologies might be of limited added value in soft tissue repair. So far, there are no human studies and very few animal studies supporting TE-based products to repair abdominal wall defects.[78,84,85] Considering the complexity of this technology and the limited number of cases where the added value will be significant, one can suspect that the demonstration of cost-effectiveness of such approach will be a challenge.

Conclusion

Despite the introduction of meshes more than 50 years ago, and large research areas, there is still room for improvement in reinforcement materials for soft tissue repair. Long-term tolerance, pain reduction, and infection resistance will remain the most important drivers for further developments. As seen, many parameters are involved for a successful repair, with some of them difficult to combine. It seems unlikely that only one mesh will fit all the indications and procedures. The meshes of the future will be specific to each main indication, surgical techniques, and maybe also specific to certain identified risk factors. The probable future profile of such meshes should be the following: highly porous; hydrophilic with potentially complementary coatings to limit classical complications such as infections or chronic inflammations; conformable and mechanically adapted to the receiving sites; shaped to the anatomy and in a form facilitating the reproducibility of the surgical technique; stable in the long term; able to be rapidly integrated and in the meantime able to preserve the noble structures present in the vicinity such as nerves, larger vessels, spermatic cords, and hollow organs. To successfully develop such a device, a multidisciplinary approach will be required, taking in account at the same time the host biology –which is to be better understood, the mesh design and structure, the surface and bulk chemical properties, and also the mechanical environment and the specificity of the clinical conditions.

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Internal Fixation and Soft-Tissue Anchors for Prolapse Repair

26

G. Willy Davila

Advances in prolapse surgery have led to the development of less invasive approaches through small incisions and minimal dissection. Most innovative procedures at this time require passage of long needles in order to direct the placement of the reconstructive mesh and creation of supporting neo-ligaments. This is true for prolapse repair kits as well as anti-incontinence slings. At least for prolapse kits, patients frequently complain of more pain from the perianal incisions required for passage of the placement needles than from the vaginal incisions themselves.

Only recently the concept of internal fixation of mesh and mesh strips has received the attention of surgical kit manufacturing companies and pelvic reconstructive surgeons. This concept follows along the commonly accepted approaches used by orthopedic surgeons for fracture and joint repairs.

Factors favoring the development of internal fixation anchors and sutures include:

1. Growing popularity of existing kits and slings using long needles
2. Recognition of sturdy anatomic supporting structures and ligaments for re-supporting prolapsed segments
3. Knowledge regarding safe and well-tolerated implantable mesh and biologic graft materials
4. Interest in making minimally invasive procedures even less invasive
5. Desire to minimize postoperative pain, morbidity, and hospitalization length of stay.

As a consequence, companies and reconstructive surgeons have developed tools to facilitate vaginal reconstruction through the incisions used during primary dissection. These tools have included:

1. Internal fixation fasteners and soft tissue anchors and anchoring systems
2. Self-anchoring sutures which do not require knots
3. Tissue fixation systems for internal anterior and posterior prolapse repair

Why Switch to Internal Fixation?

There are many reasons surgeons should be attracted to internal fixation of tissue and grafts. Top among these reasons is likely the desire to reduce the number and size of skin incisions. Consequences of reduced skin incisions include less pain, especially at dependent pressure sites where needle passage incisions are made. There is also a reduced likelihood of skin infections and cellulitis, and reduced blood loss.

It is likely possible to obtain safer and more robust anchoring to supporting structures via placement of anchoring materials through internal dissection into potential anatomic spaces toward the target structures such as sacrospinous ligaments and pelvic fascial condensation sites. Using long transcutaneously placed needles require clear 3-D mental imaging on the surgeon's part regarding the course the needle will take in reaching the target structure. Many of the complications reported in the use of the currently available kits include avoidable trauma to other pelvic structures, such as the colon and bladder, due to divergence of the needle from its intended and predetermined path. Of equal importance is the requirement that sturdy fixation to the supporting structures be achieved for appropriate prolapse repair. We frequently see apical prolapse recurrence due to detachment of the supporting mesh arms from the sacrospinous ligament, likely due to suboptimal fixation. Direct firm fixation into the sacrospinous ligament would likely be more predictably achieved if the operating surgeon is able to directly palpate the ligament he/she wishes to anchor into and thus place the soft tissue anchor firmly into this structure. Many surgeons operate primarily via palpatory guidance, and internal fixation will certainly facilitate this process. Direct placement

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will certainly be more accurate than "blind" passage through a distance of 8 or greater cm.

Use of soft tissue anchors will also reduce the dependence on sutures and knot-tying, and reduce the volume of mesh required for reconstruction by minimizing the length of mesh arms. Suture knots are associated with pain at knot sites, graft detachment if knots become "unknotted," development of granulation tissue at skin closure sites, and possible infection of the suture if multifilament permanent sutures are used. Thus, many currently avoidable technical problems could be minimized by the implementation of internal fixation techniques.

Internal Fixation Fasteners and Soft Tissue Anchors and Anchoring Systems

The first studied soft tissue fastener or anchor was developed and studied in Israel – EndoFast Reliant System (Endogun Medical Systems, Israel – Fig. 26.1). The Spider Fasteners can be deployed through vaginal incisions to fixate surgical mesh using a disposable deployment device. The fasteners are deployed through the mesh into the underlying supporting structure. In a series of 20 patients, 32 prolapse (12 anterior and 20 posterior) repairs were performed using the EndoFast system to attach mesh to soft tissue at the level of the ischial spines, ischiopubic ramus, and/or puborectalis muscles depending on the compartment being suspended. At 1 year follow-up, 83.3% of patients had optimal prolapse correction. Only one device-related complication occurred, and patient and surgeon satisfaction was high.[1,2]

In an animal model, the EndoFast system was used to test the strength of attachment of a mesh arm with the fasteners as compared to suture fixation into subcutaneous pockets or tunnels. Pull out strength as tested with a tensiometer was 8–16× greater with fastener attachment during the initial 3–7 days after implantation (1,600 vs 112–282 gr). By 15 days, all mesh arms were equally strongly attached (all around 4,000 gr).[3]

Internal fixation of suburethral slings for stress incontinence was initiated with the TVT-Secure (Gynecare) and MiniArc (American Medical Systems) in 2007. The TVT-S system relies on self-fixation of implanted polypropylene mesh once the impregnated collagen material has been broken down (Fig. 26.2a). Initially reported success rates ranged from 60% to 80% with short-term follow-up.[4] It is unclear whether these lower than expected success rates were due to the weakened soft tissue fixation or based on the technique used for implantation: retropubic "V" placement versus transobturator "U" placement. Work is under way to clarify this issue.

The Mini-Arc soft tissue anchor system is designed for direct anchoring into soft tissue, ligaments, or fascia (Fig. 26.2b). Initial reports demonstrated improved success rates, but did not approach those achieved with retropubic and transobturator slings.[5] Work is also under way to clarify factors which may optimize successful outcomes including patient selection, implantation technique, and degree of urethral intrinsic sphincteric deficiency.

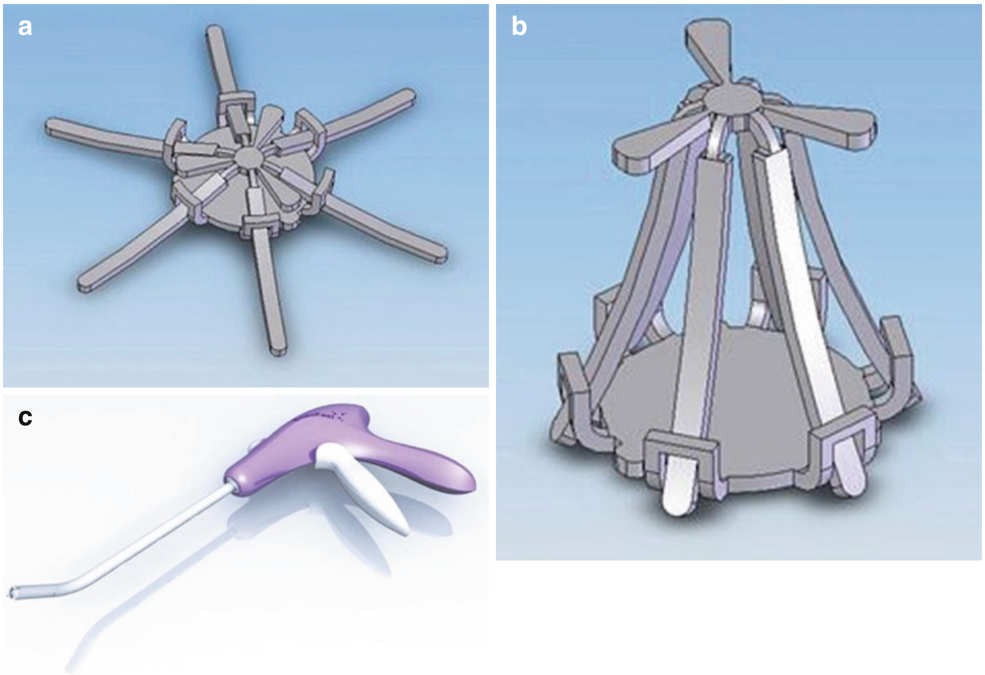


Fig. 26.1 EndoFast Reliant System for soft tissue fixation: soft tissue Spider anchor (**a**, **b**) and deployment device (**c**) (Courtesy of Endogun Medical Systems)

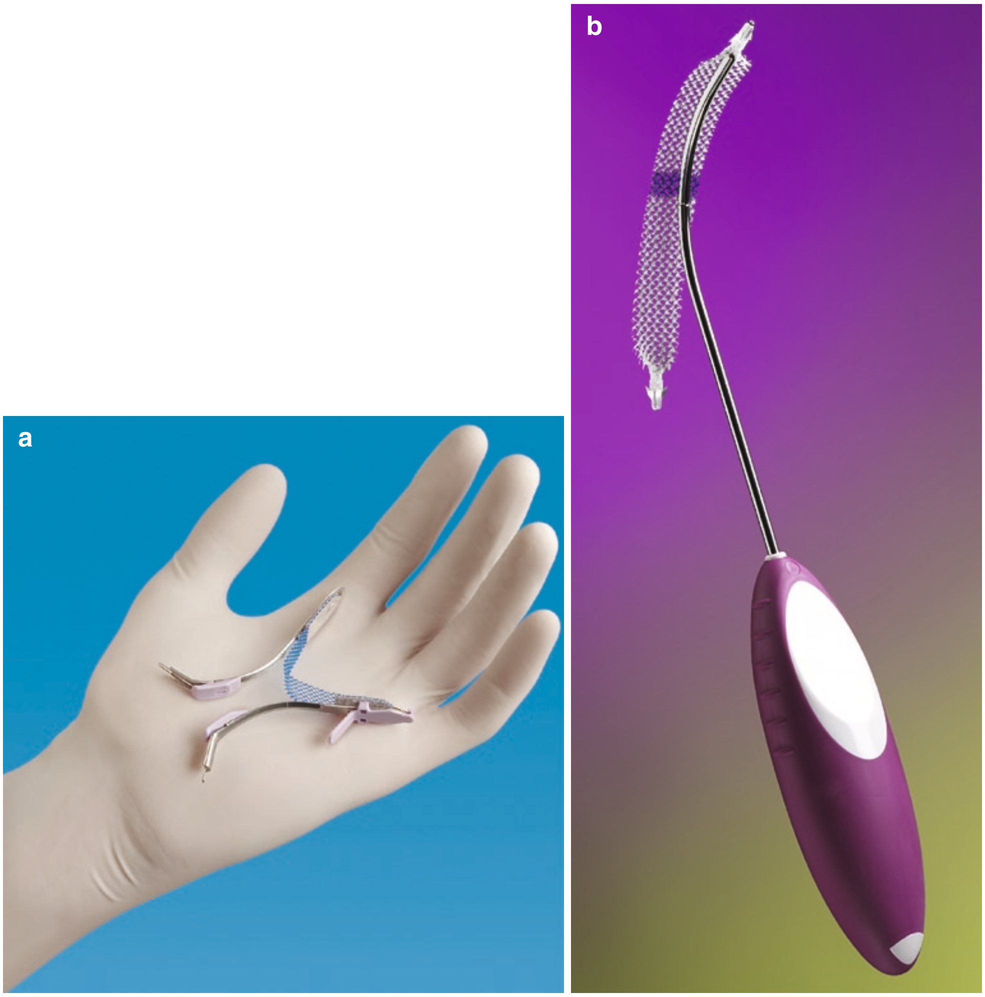


Fig. 26.2 Internal fixation slings for stress incontinence: (**a**) TVT-S (Gynecare TVT Secur System Tension-Free Support for Incontinence, © ETHICON, Inc. Reproduced with permission); and (**b**) MiniArc (Courtesy of American Medical Systems®, Inc. Minnetonka, Minnesota, www.AmericanMedicalSystems.com)

In the meantime, this soft tissue anchoring system has been expanded to use for vaginal apical prolapse. Using the same anchor mechanism to anchor mesh to the sacrospinous ligament via internal fixation has led to the development of the Elevate Vault Suspension System (American Medical Systems) via either an anterior vaginal wall dissection or a more traditional posterior wall dissection (Fig. 26.3). To date, early data with this system has demonstrated satisfactory apical suspension results.[6]

Cadaveric studies have been performed to evaluate the strength of attachment of this soft tissue anchor to various suspensory structures in the pelvis.[7] Using five cadavers, 23 anchoring sites on nine anatomical structures were selected for anchoring and testing of pullout strength using a tensiometer. In this model, the sacrospinous ligaments and Cooper's ligaments had the highest pullout strengths. However, multiple other structures were noted to have pullout strengths greater than that force resultant from a typical Valsalva effort or cough after implantation in a normal female subject – which is calculated to be no greater than 4 lb (Fig. 26.4 and Table 26.1).[8,9]

Future pelvic applications of soft tissue anchoring systems are likely forthcoming, including use in expanded sling attachment sites, alternate mesh attachment sites for prolapse repair and possibly sacrocolpopexy procedures.

Self-anchoring Sutures Which Do Not Require Knots

It is currently quite ironic as one walks through a modern operating suite that in one operating room a surgeon is placing and tying sutures by hand in a traditional unchanged approach, while in the next room a surgeon is operating using a very sophisticated highly technological robot. Until recently, using sutures meant that knots were going to be needed.

The recent advent of self-fixating barbed sutures (Quill self-retaining system, Angiotech Pharmaceuticals, Inc., Vancouver, BC, Canada), which self-fixate after placement has obviated the need to tie knots (Fig. 26.5). Applications of

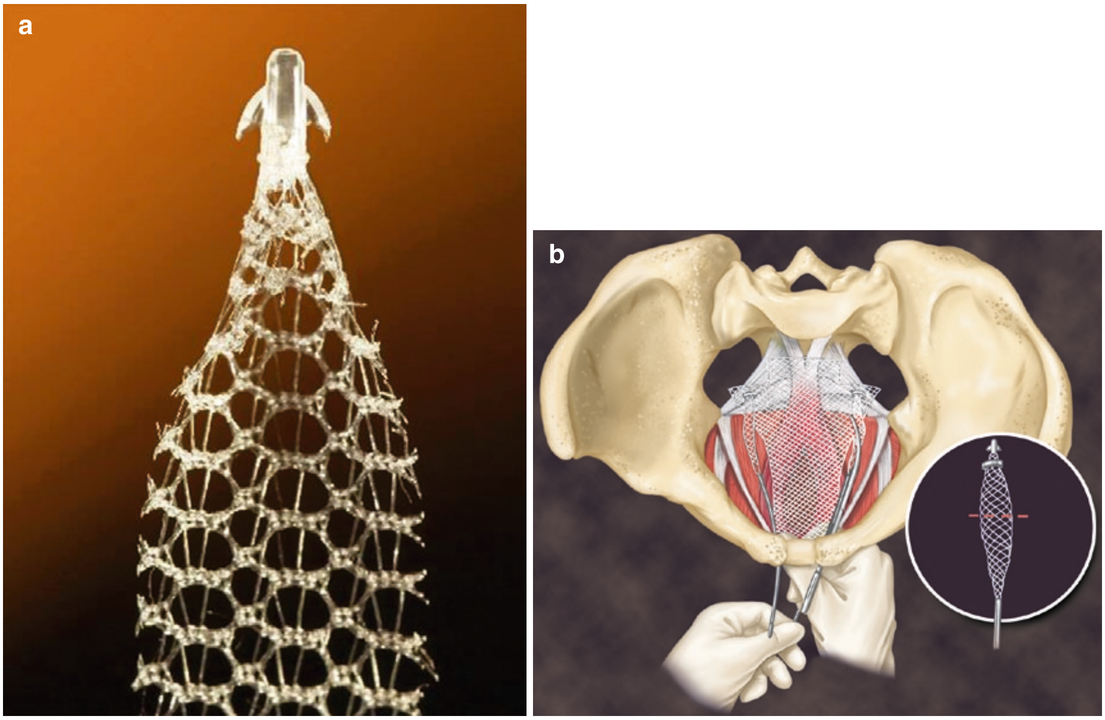
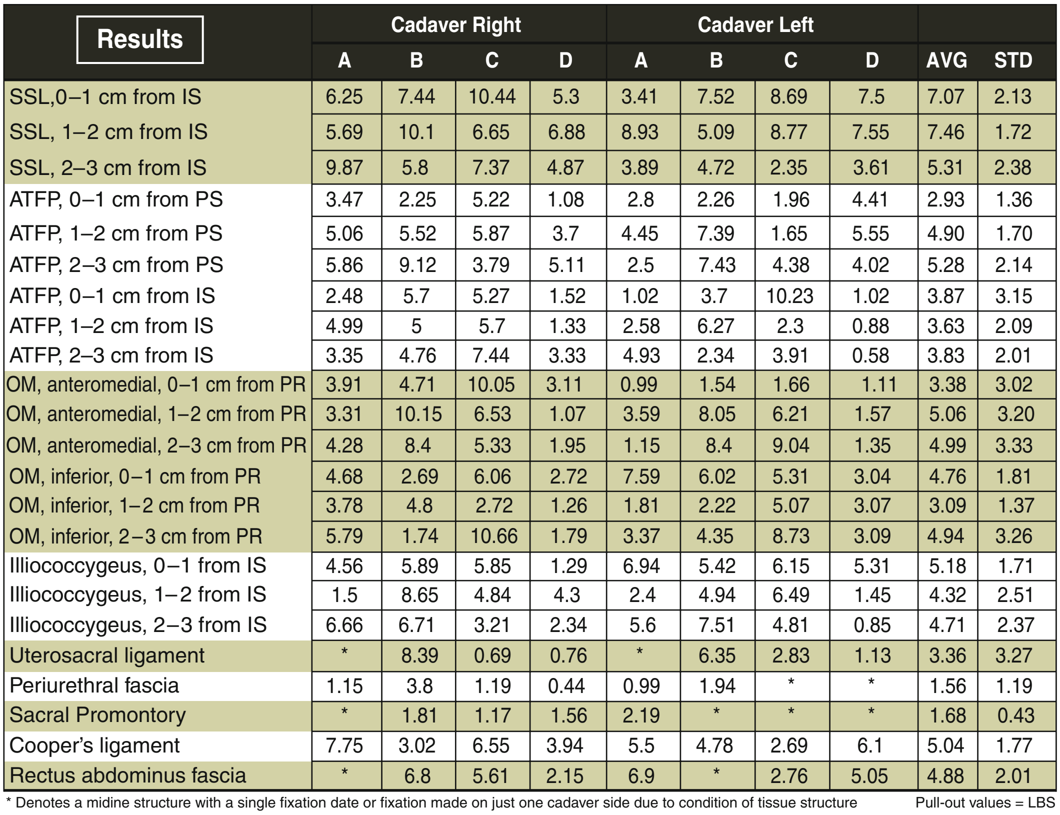


Fig. 26.3 (**a**, **b**) Elevate System for vaginal vault suspension uses two soft tissue anchors to attach arms to the sacrospinous ligaments (Courtesy of American Medical Systems®, Inc. Minnetonka, Minnesota, www.AmericanMedicalSystems.com)

Fig. 26.4 Pullout strength of MiniArc soft tissue anchor (Lbs.) when placed into various pelvic structures[7]



Results	Cadaver Right				Cadaver Left					
	A	B	C	D	A	B	C	D	AVG	STD
SSL,0–1 cm from IS	6.25	7.44	10.44	5.3	3.41	7.52	8.69	7.5	7.07	2.13
SSL, 1–2 cm from IS	5.69	10.1	6.65	6.88	8.93	5.09	8.77	7.55	7.46	1.72
SSL, 2–3 cm from IS	9.87	5.8	7.37	4.87	3.89	4.72	2.35	3.61	5.31	2.38
ATFP, 0–1 cm from PS	3.47	2.25	5.22	1.08	2.8	2.26	1.96	4.41	2.93	1.36
ATFP, 1–2 cm from PS	5.06	5.52	5.87	3.7	4.45	7.39	1.65	5.55	4.90	1.70
ATFP, 2–3 cm from PS	5.86	9.12	3.79	5.11	2.5	7.43	4.38	4.02	5.28	2.14
ATFP, 0–1 cm from IS	2.48	5.7	5.27	1.52	1.02	3.7	10.23	1.02	3.87	3.15
ATFP, 1–2 cm from IS	4.99	5	5.7	1.33	2.58	6.27	2.3	0.88	3.63	2.09
ATFP, 2–3 cm from IS	3.35	4.76	7.44	3.33	4.93	2.34	3.91	0.58	3.83	2.01
OM, anteromedial, 0–1 cm from PR	3.91	4.71	10.05	3.11	0.99	1.54	1.66	1.11	3.38	3.02
OM, anteromedial, 1–2 cm from PR	3.31	10.15	6.53	1.07	3.59	8.05	6.21	1.57	5.06	3.20
OM, anteromedial, 2–3 cm from PR	4.28	8.4	5.33	1.95	1.15	8.4	9.04	1.35	4.99	3.33
OM, inferior, 0–1 cm from PR	4.68	2.69	6.06	2.72	7.59	6.02	5.31	3.04	4.76	1.81
OM, inferior, 1–2 cm from PR	3.78	4.8	2.72	1.26	1.81	2.22	5.07	3.07	3.09	1.37
OM, inferior, 2–3 cm from PR	5.79	1.74	10.66	1.79	3.37	4.35	8.73	3.09	4.94	3.26
Illiococcygeus, 0–1 from IS	4.56	5.89	5.85	1.29	6.94	5.42	6.15	5.31	5.18	1.71
Illiococcygeus, 1–2 from IS	1.5	8.65	4.84	4.3	2.4	4.94	6.49	1.45	4.32	2.51
Illiococcygeus, 2–3 from IS	6.66	6.71	3.21	2.34	5.6	7.51	4.81	0.85	4.71	2.37
Uterosacral ligament	*	8.39	0.69	0.76	*	6.35	2.83	1.13	3.36	3.27
Periurethral fascia	1.15	3.8	1.19	0.44	0.99	1.94	*	*	1.56	1.19
Sacral Promontory	*	1.81	1.17	1.56	2.19	*	*	*	1.68	0.43
Cooper's ligament	7.75	3.02	6.55	3.94	5.5	4.78	2.69	6.1	5.04	1.77
Rectus abdominus fascia	*	6.8	5.61	2.15	6.9	*	2.76	5.05	4.88	2.01

* Denotes a midine structure with a single fixation date or fixation made on just one cadaver side due to condition of tissue structure

Pull-out values = LBS

Table 26.1 A cadaveric model for determing soft tissue fixation strength for pelvic reconstructive surgery (Reprinted from Lukban et al.[6] With permission)

Overall strength	Mean per structure (lb)
SSL	6.56
ATFP	4.38
OM	4.47
IL	5.57
USL	7.37
PUF	1.55
SP	3.75
CL	4.93
RAF	6.10

SSL sacrospinous ligament, *ATFP* arcus tendineus fascia pelvis, *OM* obturator membrane, *IL* iliococcygeus muscle, *USL* uterosacral ligament, *PUF* pubo-urethral fascia, *SP* sacral promontory, *CL* Cooper's ligament, *RAE* rectus abdominis fascia

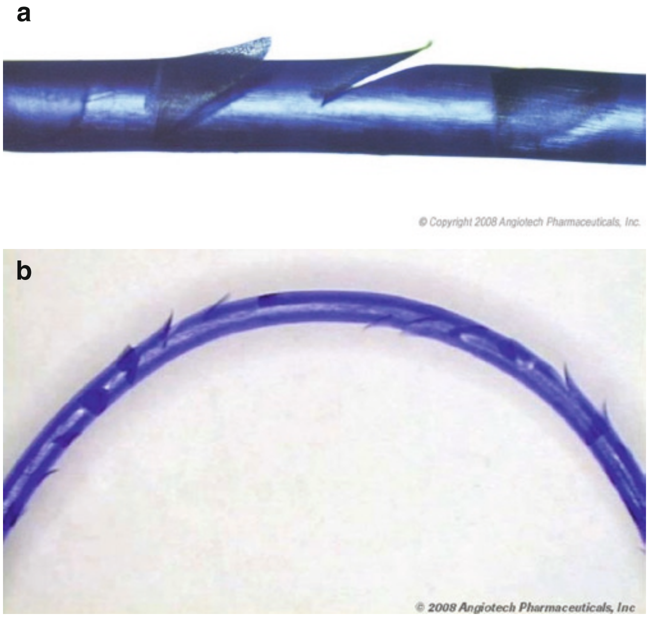

a
© Copyright 2008 Angiotech Pharmaceuticals, Inc.
b
© 2008 Angiotech Pharmaceuticals, Inc

Fig. 26.5 (**a**, **b**) Quill sutures are barbed and self-fixate, not requiring knot tying (© Angiotech Pharmaceuticals, Inc. Reprinted with permission)

self-fixating sutures include plastics, urological, and gynecological surgical procedures.[10–13] These sutures are available in multiple materials including polypropylene (Prolene), nylon, and polydioxanone (PDS) allowing for usage in multiple applications in the pelvis and elsewhere. Clinical evaluations to date have demonstrated ease of use and significant time savings, especially with laparoscopic applications. We have used these sutures for midline fascial plication cystocele repairs and vaginal skin closure, with results apparently identical to traditional techniques. Multiple clinical trials are currently underway to clarify the applicability of these sutures.

Available Adjustable Internal Fixation Systems for Anterior and Posterior Prolapse Repair

The Tissue Fixation System (TFS) comprises two small polypropylene soft tissue anchors connected to an adjustable polypropylene tape. It has been used for uterine/vault prolapse as well as anterior/cystocele repairs.[14,15] The reported series include 67 patients with uterine/vault prolapse where the tape was secured laterally to the uterosacral ligaments, and 90 patients where the TFS system was used for cystocele repair by placement of one to three tapes from pelvic sidewall to pelvic sidewall in order to reestablish anterior wall support. These preliminary reports demonstrated satisfactory prolapse repair in most subjects, with no device-related complications. When used alone, apical support tapes led to the development of cystocele in 18% of subjects. However, the authors reported a procedure length of only 5–10 min.

This system is currently not available in the USA and there is limited international experience. Thus, more confirmatory data is clearly needed. Internal fixation to the sacrospinous ligaments bilaterally in order to elevate the vaginal apex and/or uterus is the goal of this procedure. Clinical trials are currently under way, but preliminary clinical experience appears to be satisfactory.

Summary

New technological developments have led to techniques, which allow for repair of vaginal prolapse through the same vaginal dissection incisions by using internal tissue and mesh fixation as well as self-retaining sutures. There are multiple potential benefits to patients, including reduced operative time, reduced blood loss and pain. The future will certainly lead to further simplification of reconstructive techniques.

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Future Challenges

27

Peter E. Petros

Study the past if you would define the future.

Confucius

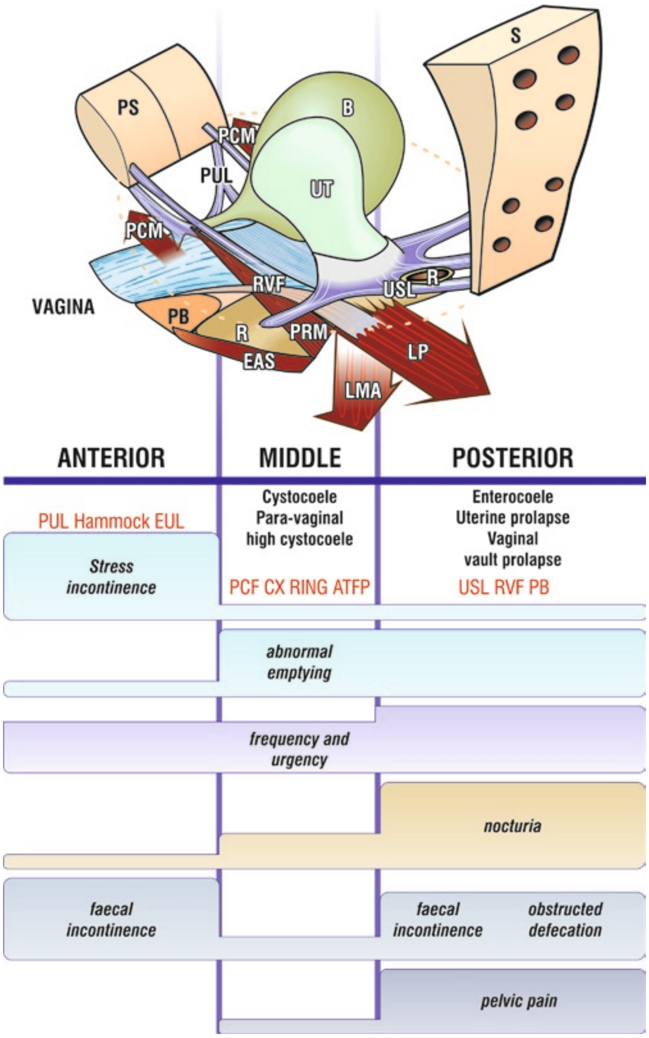
Introduction

Stress incontinence aside, it is remarkable that the focus of pelvic floor surgeons in the past, even the recent past, has been on prolapse surgery. Surgery for pelvic floor dysfunction, abnormal symptoms, has been largely neglected. Yet the population is aging, and up to 50% of admissions to Nursing Homes are for urinary/fecal incontinence and evacuation disorders. These are far greater problems for the community than organ prolapse. Even in younger women, urge incontinence, nocturia, pelvic pain, vulvodynia, interstitial cystitis can have a major impact on quality of life for individuals who have these conditions.

Fundamental to any speculation on future directions is the understanding that the pelvic floor functions as a balanced, interrelated system – "pelvic floor homeostasis."

The pelvic floor is a neurologically controlled system of organs, muscles, ligaments, nerves, and blood vessels. Each of these affects the other, and the whole, resulting in a *balanced system of pelvic floor homeostasis*.

Figure 27.1 pictorially summarizes, in a very simplistic way, this relationship between organs, muscle forces, and ligaments. It serves as a working document for now. It is also a diagnostic tool, which guides minimally invasive surgery based on the Integral Theory System – precise placement of plastic tapes to reinforce damaged suspensory ligaments.[1] These tapes work by creating artificial collagenous suspensory neoligaments, which act as anchoring points for the


PS
PCM
PUL
B
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S
PCM
VAGINA
PB
RVF
USL
R
R
PRM
EAS
LP
LMA
ANTERIOR
MIDDLE
POSTERIOR
PUL Hammock EUL
Stress incontinence
Cystocoele Para-vaginal high cystocoele
PCF CX RING ATFP
Enterocoele Uterine prolapse Vaginal vault prolapse
USL RVF PB
abnormal emptying
frequency and urgency
nocturia
faecal incontinence
faecal incontinence
obstructed defecation
pelvic pain

Fig. 27.1 The Pictorial Diagnostic Algorithm summarizes the relationships between structural damage (prolapse) in the three zones and function (symptoms). The size of the bar gives an approximate indication of the prevalence (probability) of the symptom. Laxities (red lettering) which can be repaired: pubourethral ligament (PUL); external urethral ligament (EUL); pubocervical fascia (PCF); CX ring/cardinal ligament; arcus tendineus fascia pelvis (ATFP); uterosacral ligament (USL); rectovaginal fascia (RVF); perineal body (PB).

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three directional muscle forces. Utilizing this diagnostic/surgical system, up to an 80% cure rate has been achieved for the symptoms displayed in the algorithm[2–4] (see chapter 2). And for the 20% where the surgery failed? For them, the cure rate was 0%. Herein lies the challenge for the future, how to improve existing diagnostic and surgical systems, and to extend them to conditions which have a severe impact on women's quality of life. A greater understanding of the dynamic anatomy, the interaction of the structures, and what the surgery does to the anatomy will be required before any substantive progress occurs in this type of surgery.

Anatomy

All existing pelvic floor surgery works by creating some type of scar tissue collagen. The natural suspensory ligaments such as pubourethral and uterosacral, so simplistically presented diagramatically, are very complex structures indeed. They contain collagen, smooth muscle, elastin, nerve, and blood vessels. They are an active neurologically controlled anchoring structure for the muscle forces, not just the passive collagenous strut provided by an implanted tape. The pubocervical and rectovaginal fascia have identical structural components, indicating that they too are active contractile (and expansile) structures. The stretch receptors are a complex structure, composed of many different transient receptor channels, nerve fibers, and transmitter substances. The muscles contain spindles, which sense the tension within the muscle, and therefore of the ligaments and fascia to which the muscles are inserted. Each organ muscle and ligament has its own nerve supply, and all the anatomical elements are controlled as an interrelated system by a complex, coordinated, reflex neurological feedback control system. This means that any surgical intervention in one zone, will inevitably unbalance the system, and may cause decompensation in the other zones. It follows that any surgical intervention should be minimal, and not distort the geometry of the structures.

How Surgery May Disrupt Pelvic Floor Homeostasis

From the days of Burch Colposuspension, high rates of "de novo" enterocoele, urgency, "obstructed micturition," residual urines, and pelvic pain were reported. The newer "tension-free" slings have not been immune from reports of "de novo" symptoms. To understand the pathogenesis of "de novo" symptoms, we need to better understand the anatomy of all the elements in the pelvic floor system, and how the surgery affects their interrelationship. With reference to Fig. 2.2, it is almost impossible to conceptualize a fetal head descending through the pelvis and creating major damage to one zone of the vagina without at least partial damage to the other zones. So a more objective method for diagnosis of subclinical connective tissue damage is an important goal for the future.

Future Directions

Connective Tissue Causation

Pelvic pain, interstitial cystitis, vulvodynia, urge incontinence, nocturia, and fecal incontinence can be seriously distressing to a patient, and need to be addressed in any discussion on future directions. Many of these patients have minimal, if any, prolapse. There is growing evidence that at least some of these conditions, though peripheral neurological in origin, may be, at least, partly caused by connective tissue laxity: failure to support sensitive stretch receptors (bladder), or nerve fibers (uterosacral ligaments).[5,6]

Diagnosis

As regards diagnosis, the main challenge is to develop a valid probability assessment between the nine connective tissue structures (cf diagnostic algorithm, previous chapter), and abnormal symptoms. A computerized diagnostic system and data base would provide a first step in this quest. Anonymous transfer of pre- and postoperative data to an interactive internet website will be an important tool for continuing development of pelvic floor science as it allows for sharing of research results, general relevant information, new ideas, and collaboration on a scale previously thought unimaginable. Contributing information about the structure(s) repaired, and recording the change in symptoms and objective tests, may help to provide an improved probability rating for each structure and its contribution to a particular function or dysfunction. Transperineal ultrasound is being increasingly used to diagnose minor herniations in the vagina. This may be a useful step, but such herniations will require a strong statistical correlation to symptom causation before such ultrasound findings can be added to the decision tree for surgery. More useful may be a noninvasive method for assessing vaginal elasticity. Again, normative and pathological values need to be established before this tool can be usefully applied.

Surgery

The pelvic floor functions like a tensioned suspension bridge, with the same muscle forces providing structural strength, and function, opening and closure of each organ. Understanding the dynamic interrelationship of each component structure to pelvic floor function, and the effect of surgery on the system following repair thereof, is an impossibly difficult task. The problem is always that in a complex interrelated system, any intervention which distorts even one part of the system can be multiplied through the system. This concept is best understood as the "butterfly effect," or "Law of Unintended Consequences."

In order to try and minimize unexpected effects of surgical intervention, the author's guiding surgical principle, at all times, has been that Nature is perfect, and that any intervention must be minimal, easily reversible, and mimic Nature as much as possible. This means that the organs (uterus or vagina) must not be excised, destroyed, or displaced, that elasticity needs to be preserved, so no permanent damage is inflicted. A major concern is the uncontrolled insertion of large meshes in non-anatomical positions, with no long-term data available to assess the effects. From a purely anatomical perspective, one cannot insert a mesh into the vesicovaginal or rectovaginal space without obliterating it. These spaces allow independent movement of the organs, essential in the musculoelastic system, which constitutes the pelvic floor. The "cathedral ceiling" method for vaginal wall reinforcement, uses a different structural principle to large mesh. Discrete tapes are implanted transversely, so as not to limit the anteroposterior movement of the organs. This promises to be a significant improvement, as it avoids the organ spaces.

The ideal surgical solution (perhaps through advanced application of stem cells) would use an implanted template to re-create all components of ligaments and fascia: smooth muscle, collagen, elastin, blood vessels, and nerves. This may be possible, perhaps. A link to the spinal cord and cortical feedback control centers seems implausible, however.

Conclusion

The time has arrived for including a comprehensive pre- and postoperative symptom assessment for every patient undergoing pelvic floor surgery. Such a questionnaire, which relates specific symptoms to specific structures, is available at www.integraltheory.org.

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The Future of Pelvic Organ Prolapse (POP) Surgery

28

Peter von Theobald

Pelvic Organ Prolapse (POP) Surgery

The near future in pelvic organ prolapse (POP) surgery is certainly less and less invasive and far future probably less and less surgical, possibly requiring medical preventive treatment. In a near future, the most painful and frightening part of the vaginal operations, the perineal transfixiation, will be replaced by less aggressive techniques, fixing the meshes through the vaginal incision. The risk of vessel or nerve injury will be suppressed by avoiding the "blind way" of the tunnellers and needles. Two preclinical trials have been performed in the University Hospital of Caen: one using a new fastener (Endofast™) and the other using fibrine glue (Tissucol™).

The Endofast Reliant™ System

A prospective multicenter study was performed to evaluate the efficacy and safety of EndoFast Reliant™ system for POP repair. (Menachem Alcalay from Chaim Sheba Medical Center, Israel, Michel Cosson and Jean-Philippe Lucot from CHU Lille, France, Peter von Theobald from CHU Caen, France). The EndoFast Reliant™ system is a new minimally invasive transvaginal technique for pelvic organ prolapse (POP) repair. This system reinforces the pelvic floor with polypropylene mesh with soft-tissue fasteners, requiring a single intravaginal incision and avoiding the use of trocars (see Fig. 26.1).

Study Design

Between March and November 2007, a prospective multicenter study was carried out in 20 women with anterior and/or posterior POP, who underwent vaginal repairs with mesh reinforcement. The fasteners anchored the mesh into the soft tissue, adjacent to the ischial spines and posterior symphysis for the anterior compartment and at the ischial spines and puborectalis for the posterior compartment. Eleven patients (79%) underwent double compartment corrections.

The study was approved by the institutional research board at each center and all patients signed informed consent to be included. We excluded patients who needed hysterectomy or correction of stress urinary incontinence. All patients had thorough evaluation including physical examination (using the POP-Q system), pelvic floor symptom evaluation using the pelvic floor distress inventory (PFDI) questionnaire and sexual function assessment using the FSFI questionnaire. Following surgery, the physician's satisfaction was documented. The patients were followed at 2 weeks, 3 and 6 months postoperatively, using the same measures that were evaluated at the preoperative visit. To follow possible migration of the fasteners, the patients had X-ray of the bony pelvis after the procedure and following 3 months. For the statistical analysis we used SAS software.

Results

The surgical procedure was performed under general or regional anesthesia. To date, all patients reached 3-month follow-up and 12 patients had 6-month follow up. Mean age was 61.2 years (range: 34.2–79.2) and the mean BMI was 25.9 (range: 21.6–29.0). Twelve (60%) out of 20 patients had double compartment (Anterior & Posterior) correction. There were no intraoperative complications and up to discharge, no major complications were observed. At 3-months follow-up: one fastener misplacement was noticed, picking through the vaginal mucosa. The fastener was duly removed under local anesthesia with no clinical consequences after 1 year. One non-symptomatic wound dehiscence was reported, without mesh exposure and treated uneventfully. Neither mesh erosion nor fastener migration was noticed. Two cases of de novo SUI occurred (10%), of whom one was treated surgically. One case of misplacement of a single fastener was observed due to dyspareunia and removed under local anesthesia.

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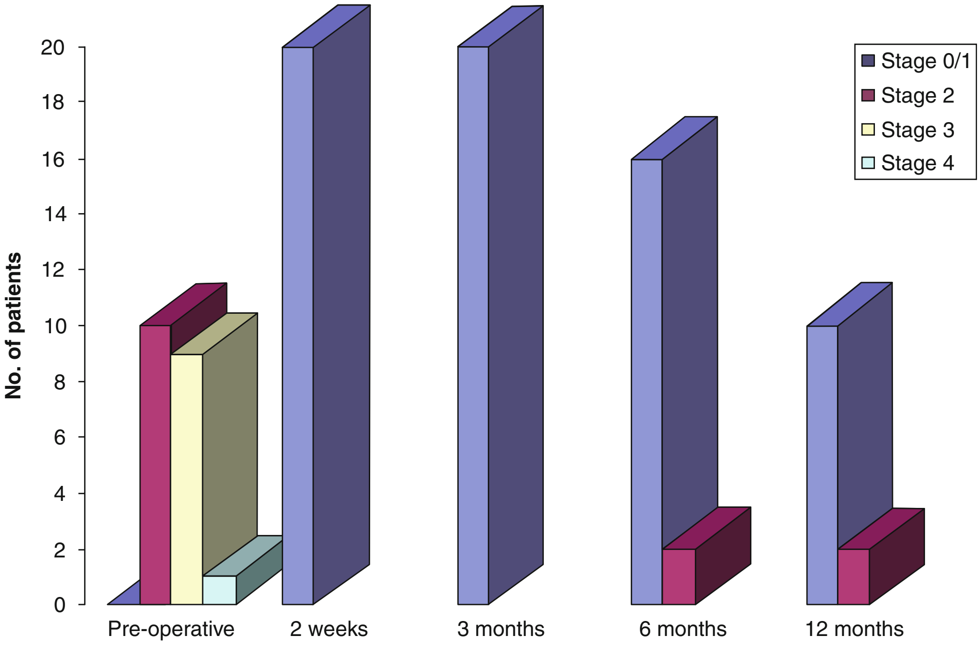
Fig. 28.1 POPQ prolapse grading pre- and post-procedure. In the 32 procedures performed in 20 patients, prolapse correction has been in general maintained during the follow-up period, as shown by the POP-Q measures performed during the follow-up visits (At 3 months, 6 months, and 1 year where follow-up is available)



Prolapse in clinical examination resolved in 100% of our patients (Fig. 28.1, Table 28.1) at 3 months (Grade 0 or 1) and in 86.7% at 6 months (2 patients out of 15 had grade 2 prolapse). Consistent improvement in pelvic floor symptoms related to prolapse bladder or rectum, and POP-Q measures are shown in (mean ± SD) (Table 28.2).

To date, nine patients are practicing sexual intercourse postoperatively: three patients who were previously abstinent started practicing sexual intercourse following the procedure. Satisfaction during intercourse, as measured by FSFI questionnaire, has improved from a mean score of 1.3–2.9 ($p = 0.032$). Pain during intercourse decreased significantly after the procedure from a mean score of 2.6 at screening to a mean score of 4.9 at 6 months ($p = 0.006$). Ratings of the physician's feedback from the procedure were high, regarding satisfaction from the procedure (very satisfied: 63.2%; somewhat satisfied: 31.6%) and safety assessment (very satisfied: 73.7%; somewhat satisfied: 26.3%). We conclude that this system is an attractive and safe option for mesh use during pelvic organ prolapse repair, though longer follow-up is needed.

The Tisspro Protocol Using Fibrine Glue (Tissucol)*

The Tisspro Protocol using fibrine glue (Tissucol*), a preliminary study, was performed in our center, including 11 patients between November 2004 and May 2006. Table 28.3 shows the procedures performed on these patients. Figures 28.2 and 28.3 show the special design of the meshes to allow gluing to the iliococcygeus and obturator internis muscles.

Operative Protocol

The paravesical and pararectal dissections are identical to the previously described mesh techniques. The meshes are well spread in the right position: for the posterior mesh, the lateral surfaces lying on the iliococcygeus muscle and the sacrospinous ligament, for the anterior mesh, on the obturator internis muscle and on the arcus tendineous elevator ani. The Tissucol* fibrin glue is then sprayed on the mesh and the

Table 28.1 POP quantification changes over time for 3, 6, and 12-month follow-up

	Ba	Bp	C	D
Baseline	0.8 + 1.6	−1.6 + 1.8	−2.6 + 4.1	−4.5 + 2.8
3 months FU	−2.5 + 0.8	−2.8 + 0.5	−7.2 + 1.2	−8.2 + 0.9
6 months FU	−2.4 + 1.1	−2.4 + 1.7	−6.7 + 2.1	−8.3 + 0.9
	$p < 0.001$	$p = 0.206$	$p < 0.001$	$p < 0.001$
12 months FU	−2.3 + 1.4	−3.0 + 0.0	−6.6 + 2.5	−7.8 + 1.8
	$p < 0.001$	$p = 0.052$	$p < 0.001$	$p < 0.001$

Measures in centimeter from the hymeneal line, positive if out of the vagina, negative if in the vagina, Ba for the cystocele, Bp for the rectocele, C for the uterine prolapse, D for the enterocele

Table 28.2 PFDI SF–20 symptoms scoring consistent improvement in bladder symptoms related to prolapse and a significant improvement in prolapse symptoms were observed by PFDI measures

	Bladder symptoms	Anorectal symptoms	Prolapse symptoms
Baseline	1.4 ± 1.6	0.3 ± 0.6	4.1 ± 1.5
3 months FU	0.6 ± 1.0	0.2 ± 0.5	0.6 ± 1.2
6 months FU	0.5 ± 0.8	0.2 ± 0.5	0.8 ± 2.1
12 months FU	0.2 ± 0.4	0.3 ± 0.9	0.1 ± 0.3
	$p = 0.067$	(p = NS)	p < 0.001

Table 28.3 Listing of the 11 patients included and the procedures

N°	Cystocele	Vault	Rectocele
1	spm TO	ivs	Tissucol + spm
2	Tissucol + spm	ivs	Tissucol + spm
3	Tissucol + ugytex	ivs	Tissucol + ugytex
4	Tissucol + spm	Obtape ivs	Tissucol + spm
5	Tissucol + spm	Obtape ivs	Tissucol + spm
6	Tissucol + spm	Tissucol	Tissucol + spm
7	Tissucol + spm	Tissuco + Hystectl	Tissucol + spm
8	Tissucol + spm	Tissucol	Tissucol + spm
9	0	Tissucol	Tissucol + spm
10	Tissucol + spm	0	0
11	Tissucol + spm	Tissucol	Tissucol + spm

spm Surgipromesh, *TO* transobturator, *ivs* Intravaginal slingplasty

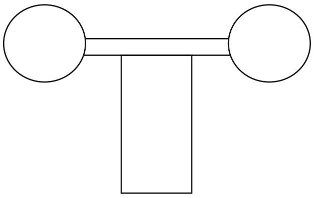


Fig. 28.2 Shape of the posterior mesh for level 1 and rectocele repair using fibrin glue

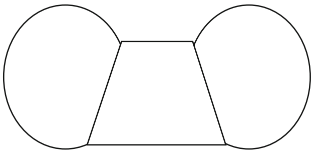


Fig. 28.3 Shape of the anterior mesh for cystocele repair using fibrin glue

tissue is held firmly in correct position during 3 min. The mucosa is sutured with a quick absorbable suture.

The anatomical results were satisfying as shown in Figs. 28.4 and 28.5. One patient presented an erosion of the anterior mesh at 6-month control. The mesh was removed easily, having no translevator arms. No other per or postoperative complication occurred. Pain scores were very low in the two postoperative days at the hospital (EVA < 3).

This preliminary study raised the interest of this innovative microinvasive technique using the adhesive effect of Tissucol*, already used in abdominal wall defect surgery for the fixation of meshes.[1,2] A multicentric prospective trial has been started in France in September 2008, including 100 patients in three centers and approved by the institutional research committee. Its aims are to test the feasibility and reproducibility of this technique as well as the effectiveness and the associated morbidity.

Concerning the Far Future

The precise role of estrogens in the pathogenesis of the pelvic organ prolapse is still unknown but as in the bone, the proteic matrix of the connective tissue of the pelvic floor is influenced by estrogens via two different receptors, alpha and beta. Selective estrogen receptor modulators (SERM), commonly used for osteoporosis, seem to have an impact on the collagen tissue of the pelvic floor. The first study, drawn from the MORE trial, showed a strong protective influence of raloxifene versus placebo on the development of POP after 9 months of therapy with a 50% reduction of the surgical prolapse or stress incontinence repair risk in postmenopausal women.[3] Other SERMs, such as levormeloxifene and idoxifene, appeared to worsen the prolapse when compared with conjugated equine estrogen and placebo.[4] We believe that the inevitable changes in the estrogen receptor expression during women's lifetime may affect the risk of POP progression and could be the reason of different effect of SERMs treatment in women with POP.

A preliminary study[5] was led in our department, aiming to quantify the mRNA levels of both forms of estrogen receptors α and β (ERα and ERβ) in the vesico- and rectovaginal walls of pelvic floor in relation to menopausal status and presence of POP. Sixty biopsy specimens from pelvic floor tissues were obtained from thirty patients categorized into four groups:

1. Non-menopausal women with POP (n = 4, mean age 40.7 ± 6.0 years)
2. Non-menopausal women without POP (n = 5, mean age 47.3 ± 3.0 years)
3. Postmenopausal women with POP (n = 12, mean age 62.9 ± 8.2 years)
4. Postmenopausal women without POP (n = 9, mean age 65.0 ± 12.2 years)

The quantification of the mRNA levels of estrogen receptors was carried out in samples of connective tissue obtained at the upper third of the vesico- and rectovaginal wall from patients undergoing hysterectomy or surgery for prolapse.

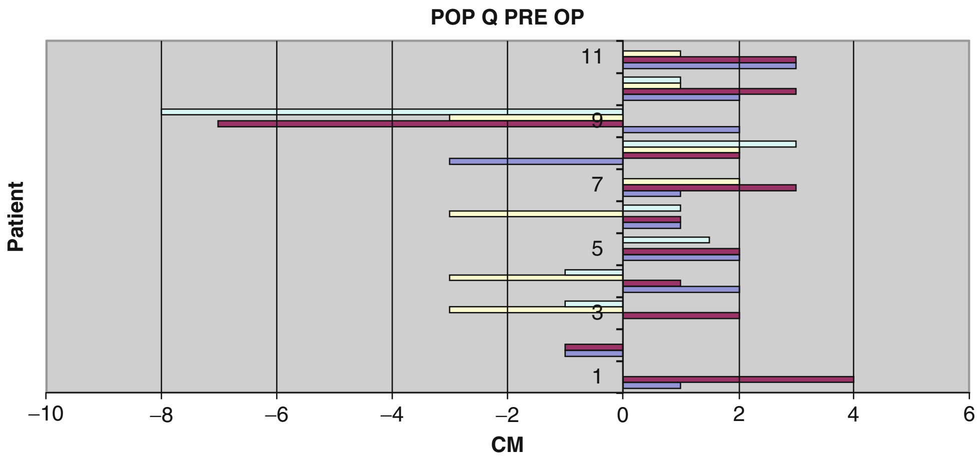


Fig. 28.4 Preoperative POP-Q scoring

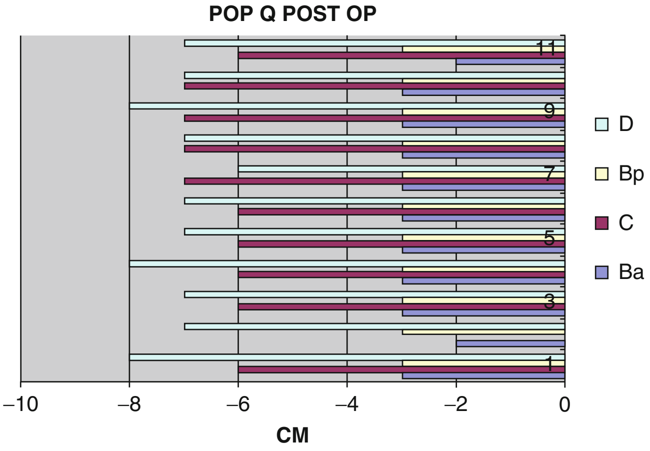


Fig. 28.5 Postoperative POP-Q scoring

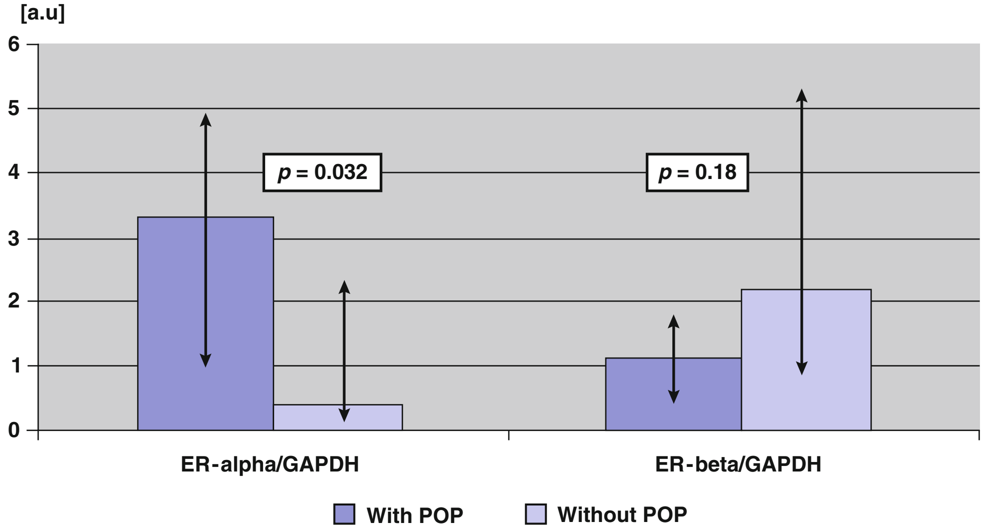
All samples were also examined by pathologists. Samples were deep frozen in liquid nitrogen and stored until RNA extraction. The RNA was isolated according to the Chomczynski's protocol. ER-α and ER-β mRNAs were measured by quantitative assays based on reverse transcription (RT) of the mRNA and real-time polymerase chain reaction (PCR) amplification of the cDNA. Using the RT-PCR technique, mRNA of both ERs was successfully detected in the tissue of fascia vesicovaginal and the fascia vaginorectal. The quantities of the studied ER transcripts were calculated using the standard curves for ERα or ERβ and normalized by the level of GAPDH mRNA and by correction factors: 10^{-2} and 10^{-3} for the ERα and ERβ expressions, respectively. Since the expression of ERα and ERβ were non-normally distributed, the studied variables were expressed as median and 1–3 quartile range. The differences between the groups were compared by Mann-Whitney U test using the Statistica 5.1 program (Statsoft 5.1, Tulsa, USA). The results were considered significant when *p* values were <0.05.

Results

In the present study the women with POP have statistically significant higher expression of ERα and higher ERα/ERβ ratio (Figs. 28.6 and 28.7). Hence, it is obvious that mRNA levels cannot precisely parallel the protein expression and reflect actual receptor status. On the other hand, the evaluation of ERα and ERβ mRNA expression levels enables the analysis of factors that were presumably implicated in the regulation of gene expression. In vitro studies showed that synthesis of ERs is regulated by SERMs and E_2. In this setting, it is reasonable to anticipate that expression of ERs varies depending on the status of menopause. The presence of estrogen receptors ERα and ERβ in connective tissues makes the pelvic floor a target for estrogens and selective estrogen receptor modulators. The remodeling of the pelvic connective tissue is likely to be concerned with aging and menopause. Collagen metabolism associated with menopause has been observed in bones and skin.[6,7] Since abnormal collagen metabolism has been observed in the vaginal tissues of women with genitourinary prolapse, the agents that positively affect collagen turnover may restore pelvic tone and reduce the incidence of pelvic floor relaxation. Although no data yet supports a remodeling effect of raloxifene in pelvic floor tissues, it is well known that raloxifene modulates collagen turnover in the skeleton. An effect that may contribute, in part, to the 30–50% reduction in risk for vertebral fracture in postmenopausal osteoporotic women on raloxifene therapy[8,9]. Our results show the different expression of estrogen receptors concerning pre- and postmenopausal women. Therefore, it makes us wonder if the observed quantitative change could participate in the pathogenesis of the development of POP.

A lot of experimental work still has to be done in this field, but if this hypothesis is confirmed, we can imagine a medical preventive treatment for POP. A SERM, maybe combined to some oral contraceptive or prescribed to high risk patients, will possibly replace our highly advanced mesh surgery!

Fig. 28.6 ERα and ERβ expression (median, 1–3 quartiles) in connective tissue of vesicovaginal fascia and rectovaginal fascia in women with POP versus without POP (all studied)



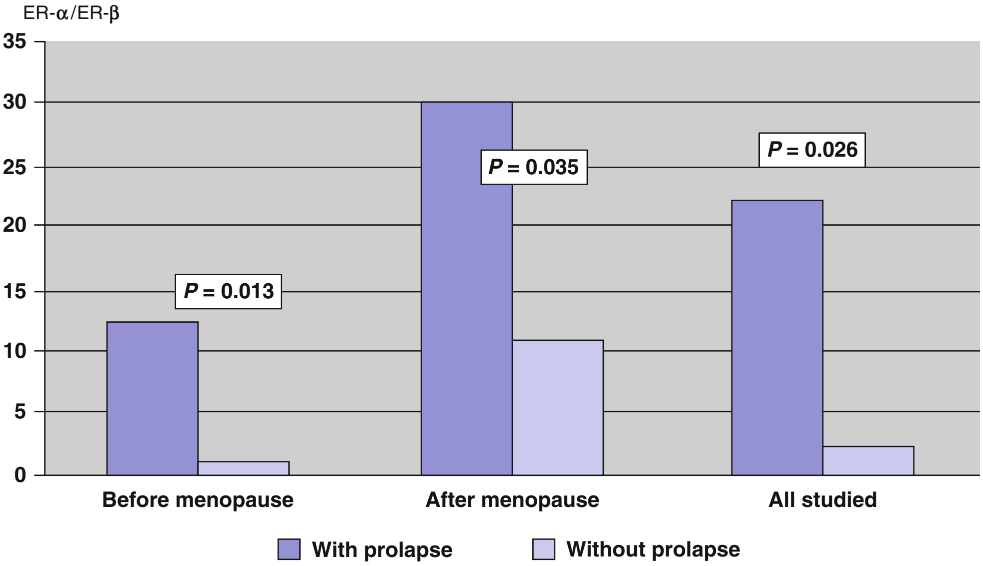


Fig. 28.7 ERα/ERβ ratio in women with POP versus without POP (before or after menopause and in all studied women)

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